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Review of Wildlife Disease Status in Game Animals in North America

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Ir

ntroduction

This review of North American game animal disease status, financed through the Agriculture Development Fund, was conducted to assemble current background information for developing disease testing protocols to govern imports of game animals to Saskatchewan. The review was encouraged by the Saskatchewan Game Farming Technical Advisory Committee (SGFTAC) which is comprised of representatives from:

Saskatchewan Game Farmers Association;

Saskatchewan Wildlife Federation;

Saskatchewan Agriculture;

Saskatchewan Parks and Renewable resources; and

Agriculture Canada.

The SGFTAC was concerned that without adequate disease testing protocols, importation of game farm breeding stock could result in the accidental introduction of new diseases. Such an occurrence could have devastating effects on the game farming industry, the province's wildlife resources, and domestic livestock industries.

Experts were contracted to review viral, bacterial and parasitic diseases of game animals of North America.

The review of each disease included:

known geographical distribution of the disease;

species affected by the disease;

method of transmission;

clinical symptoms (normal and abnormal hosts);

diagnostic procedures; and

treatment procedures.

Common diseases known to be endemic in Saskatchewan wildlife populations and not considered to present an immediate or long term threat to the game farming industry were not included in this review.

Each disease presented in this report was thoroughly discussed at a two-day conference attended by contracted experts, staff from the Western College of Veterinary Medicine,and representatives from Saskatchewan Agriculture, Saskatchewan Parks and Renewable Resources, the Saskatchewan Game Farmers Association,and the Saskatchewan Wildlife Federation. Where inadequate knowledge existed on diseases that, if introduced to Saskatchewan, would present a serious risk to the game farming industry and the provincial wildlife resource, the conference identified additional research requirements for developing new or refining existing disease testing protocols for importing game animals to Saskatchewan.

Leptospirosis

E. M. Addison
May, 1989

Disease:

Leptospirosis is an infectious spirochaetal disease of livestock and wildlife. It is also a zoonosis. It is known by a multitude of common names including Well's disease, seven-day fever, marsh fever, swamp fever, harvest fever and pea-pickers disease (see Torten 1979).

Agent:

The agents of leptospirosis are **Leptospira** spp. Leptospires are classified based on our ability to distiguish between them serologically. More than 180 distinct serovars are currently recognized (Shotts 1981). Some serovars are closely associated with particular hosts. For example, bovine leptospirosis in North America is usually caused by **canicola, grippotyphosa, hardjo, icterohaemorrhagiae, pomona, szwajizak**, but other serovars may be involved (Hanson 1977).

Leptospira spp. are fragile and are readily killed by desiccation, many disinfectants, and heat.

Geographic And Host Distributions:

Leptospires were first discovered in humans in Japan in 1914 but are now known to be worldwide in distribution. They also have a wide host distribution ranging from fish and reptiles to mammals. They have been reported infrequently from birds (see Torten 1979).

The known distribution of leptospires in wild ungulates of North America are summarized below.

White-tailed Deer

The distribution and prevalence of leptospirosis in white-tailed deer up to 1980 have been well reviewed by Shotts (1981). He cites literature with seropositive cases of leptospirosis in deer from Minnesota, Ohio, Wisconsin, Louisiana, Alabama, Kentucky, Georgia, Virginia, Maryland, Tennessee, Florida, Arkansas, Mississippi, Arizona, Indiana, North Carolina, Oklahoma, Texas, Illinois, Iowa, Nebraska, New York, Michigan, Pennsylvania, and Ontario. Prevalence varied in these studies from less than 1% to as much as 30% with prevalence in most studies of 4-10%.

Serovars represented from these studies of leptospirosis in white-tailed deer include **grippotyphosa, pomona, canicola, australis, icterohaemorrhagiae, pyrogenes, tarassovia, georgia, ballum, sejroe, bataviae**, and **autumnalis**. More recent sero surveys of white-tailed deer for leptospirosis (Fournier et al. 1986, Ingebrigtsen et al. 1986) are similar to the pattern observed in previous studies.

Moose

McGowan et al. (1963) were the first to demonstrate evidence of leptospirosis in moose with 7% of 58 moose in northwestern Ontario seropositive. The serovars, **pomono, autumnalis, hardjo**, and **biflexus** were subsequently reported from Ontario moose (Kingscote 1971). The serovars **grippotyphosa** and **pomona** have also been identified from Minnesota moose (See Anderson and tankester 1974). From Quebec, Bourque and Higgins (1984) found only one seropositive blood sample (**grippotyphosa**) out of 208 samples examined. From Alaskan moose, Grauvogel (1984) reported 13% of 86 animals seropositive, Kocan et al. (1986) reported a total of 5 serovars for a prevalence of 12%, and Zarnke (1988) reported **ballum, grippotyphosa, autumnalis, pyrogenes, australis, and hardjo** as the serovars present in 5% of 100 seropositive samples.

Other wildlife species

Other species seropositive to leptospires include muskoxen in Alaska (83%) (Zarnke 1986); caribou in Alaska (2%) (Zarnke 1983); black-tailed deer in California (100%) (Cirone et al. 1978); mule deer (0.6%), elk (0.4%) and pronghorn (12.4%) in Colorado (Adrian and Keiss 1977); pronghorn in Wyoming (0-25%) (Thome et al. 1982); elk in southwestern Albeita (Kingscote et al. 1987); bison in Alaska (0-16%) (Zamke 1983); and axis deer (3%) and fallow deer (14%) in California (Riemann et al. 1979).

Wildlife serologically negative

There are populations listed above in which there are few animals seropositive for leptospirosis. In addition, there are populations in which sera of all tested animals were negative. Examples include pronghorn (59) in Saskatchewan and Alberta (Barrett and Chalmers 1975), muskoxen (132) in Greenland (Clausen 1986), Dall's sheep (73) in Alaska (Foreyt et al. 1983:), and red deer (27) and sika deer (4) in a New Zealand study (Hathaway et al. 1981).

Domestic livestock

Leptospires are common in livestock. Cattle are known to be infected throughout Canada. Common serovars from Canadian cattle are **hardjo** and **pomona** with **grippotyphosa** being reported on occasion. Other "hardjo-like" serovars such as **sejroe, balcanica** and **szwajizak** may also be present (Kingscote 1985a). Kingscote (1985b) reported 8% of 18,010 cattle in 24% of herds in Alberta to be seropositive for **hardjo**. Evidence of **Pomona** was less prevalent (0.5%) and less widely distributed (in 2% of herds). There was a two-fold increase in the number of **hardjo** positive herds in parts of southern Alberta from 1980-82 to 1984-85 (Kingscote 1985b).

Antibodies in Canadian sheep are less frequently observed. In fact in western Canada ovine leptospirosis appears uncommon. Kingscote (1985c) recovered **pomona** and **hardjo** from 1.0% and 0.4% of western Canadian sheep sampled. However, sera from 120 lambs from Oregon revealed antibodies to **pomona**, **hardjo**, and **grippotyphosa** in 2%, 62%, and 59% of sample respectively (Kingscote 1985c).

Although horses were not considered a host for leptospires for many years, recent surveys in Ontario have demonstrated otherwise. Kitson-Piggot and Prescott (1987) recovered antibodies to **autumnalis**, **bratislava**, **canicola**, **grippotyphosa**, **harcuo**, **icterohaemorrhagiae** and **pomona** serovars. The prevalence of **bratislava** was high, being 5% in 2-3 year old horses and 52% in horses over 7 years of age. The prevalence of other serovars ranged from 1-4% for microscopic agglutination titres >= 1.80 (Yitson:Piggot and Prescott 1987).

Pathology:

Cattle

Symptoms can vary from total absence to severe icterohaemorrhagic disease. Animals may commence with fevers for 1-7 days that is usually accompanied by anoreba, dull appearance and mastitis and a drop in milk production in dairy cattle. Haemoglobinuria and anglemia may accompeuiy liaemolytic icterus which can be fatal. Abortions and birth of retarded calves can result (Torten 1979).

Goats, sheep, and buffalo

The pathology in these species can be highly variable and generally similar to that in cattle. The extent of the pathology may be influenced by the serovar involved. For example, sheep are not considered by some to be natural hosts for **pomona** and **hardjo** (Blackmore 1982). This is seen as one reason why sheep infections with these serovars are often of siblort duration and produce severe pathological effects (see Kingscote 1985c).

Swine

In swine, infection often starts with fever, anorexia and may include diarrhoea. The disease is usually mild. Haemolytic icterus rarely occurs. Abortions and birth of retarded piglets can be common.

Horses

A mild form of the disease, somewhat similar to that observed in swine, is seen in horses.

Wildlife:

Trainer et al. (1961) found experimental infection of white-tailed deer to produce unapparent infection but they did observe abortion in 4 of 5 pregnant does. Reilly et al. (1962) also found only mild disease in experimentally infected white-tailed deer. McGowan et al. (1963) isolated leptospires from the aborted fetus of a naturally infected deer.

Six deaths in a captive black-tailed deer herd have been attributed to leptospirosis (Rapley et al. 1981). One captive DaU's sheep also succumbed to leptospirosis at the same time.

Leptospirosis was provisionally diagnosed as the cause of death of 12 pronghom examined during a die-off of pronghom in Wyoming in 1966. However, leptospires could not be recovered to confirm the diagnosis (Thome et al. 1982).

Although the pathogenesis of leptospiral infections in wild ungulates is not well understood and may be variable as in some livestock, there is sufficient cause for concern.

Public Health Implications:

People working around infected livestock frequently acquire leptospires. Recorded symptoms in humans include fever, general malaise, weakness in muscles, bronchitis, bronchopneumionia, pneumonia, anaemia, haemorrhages in skin and mucosal membranes, conjunctivities, enteritis, gastroenteritis, vomiting, neckaches, headaches, hepatitis, encephalitis, meningitis, acute nephritis and more (Torten 1979)!

As with livestock, infections may be asymptomatic or highly variable in form. The incubation period is 3-20 days and the disease usually lasts 14-21 days (Toden 1979). Mortality in humans with severe jaundice may reach 10-20%.

Transmission:

Contact with contaminated water and foods are considered the most common forms of transmission although transplacental transmission also occurs (Shotts 1981). The leptospires may penetrate broken skin or mucous membranes and may possibly also enter by ingestion (Diesch and Elhnghansen 1975).

Diagnosis:

Because the disease is variable in nature and often asymptomatic diagnosis of leptospirosis cannot be based on clinical signs. Three types of methods used are demonstration, serologic, and bacteriologic methods (see Shotts 1981).

Demonstration involves examination of fresh tissues with dark-field microscopy or appropriately stained tissues prepared histologically. These techniques should be accompanied by additioned techniques.

Serologic diagnosis involves the identity of specific antigens in serum. Serum samples should be tested against a wide variety of serovars. I recommend contacting experts such as Drs. Emmett Shotts or Barbara Kingscote for specific details on the best methods to use.

The Definitive diagnosis is achieved by isolating the leptospires. Experts should be consulted on the techniques to be used.

Treatment:

There is little information regarding the treatment of wildlife for leptospirosis. During the epizootic in black-tailed deer at the Toronto zoo, Rapley et al (1981) administered Leptoferm GHP, Leptoferm C-I, antibiotics, and supportive therapy with Haemalife (iron dextran), B-complex, multiple vitamins and Bo-Se (vitamin E and selenium). Leptospiral vaccination reduced the possibility of transmission but does not arrest established infections.

The disease is best managed by preventative control techniques supplemented by treatment. Suggestions include:

1) Blood tests animals to determine prevalence on own herds. Do so on a periodic basis.
2) If possible, do not accept animals except from leptospire-free herds.
3) Treat all introduced animals in isolation.
4) Vaccinate all animals over 6-9 months of age and do so for 2-3 years but only in enzootic areas.
5) Avoid communal pasturing with potentially infected herds.
6) Submit to pathology lab all fetuses born dead.
7) Keep herds at moderate densities because this may reduce amplified transmission.
8) Drainage of wet areas of pasture will reduce possibilities of transmission from skunks, raccoons, and other wildlife.
9) Exclusion of infected bulls from breeding.
10) Use automatic water sources off the ground.

Research Required:

Much more needs to be known about the pathogenicity of leptospires in white-tailed deer and many other species of wild ungulates. Studies involving experimental infections will give a more clear understanding of the pathogenesis of livestock serovars in wild ungulates.

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Group Discussion:

Is it possible that leptospirosis may be transmitted from cattle to wildlife?

Yes there is a distinct likelihood that this can occur. Many disease specialists suspect that transmission of the leptospires from livestock to cervids occurs more frequently than from cervids to livestock. However, at least one wild animal, the skunk, is considered a possible source of leptospires for transmission to livestock. There may be other wildlife reservoirs of concern but these do not include deer in areas where extensive studies have been done.

What evidence is there of reduced production in livestock caused by leptospirosis?

Epidemiological evidence of this type is very hard to gather. An outbreak can cause losses depending on its severity. The majority of losses associated with infections with leptospires are likely abortions.

There is not a major concem about leptospirosis in livestock because its effects are considered marginal as compared to the impact of other diseases on livestock.

What additional methods of prevention and treatment are available?

An excellent preventative measure not described above, is the use of raised mechanical water supplies for livestock. This will reduce the likelihood of the livestock drinking their water from open swamps and ponds where skunks or livestock are more likely to have infected the water supply by urinating in it. This may be the single most valuable preventative measure available.

With outbreaks of leptospirosis in sheep, herds are often vaccinated.

Lyme Disease

E. M. Addison
May, 1989

Disease:

Lyme disease is a serious zoonotic disease in humans. The same disease, also known as erythema chronicum misrans (ECM) has been recognized ia humans in Europe since 1909. The disease was first recognized in North Americans in 1969 (Scrimenti 1970). Lyme disease is being recognized as a disease in other mammals. Because the agent has only been known for 6-7 years, understanding of Lyme disease is very incomplete.

Agent:

The agent of Lyme disease was identified in 1982 as the spirochaete called **Borrelia burgdorferi** (Burgdorfer et al. 1982).

Geographic Distribution:

ECM, the skin lesion characteristic of Lyme disease, was described in Sweden in 1909. The disease, as we now know, occurs in Great Britain, most of continental Europe north of the Mediterranean and east into Russia (Schmid 1985). It is apparently reported from at least 34 states in the United States (Isaacs 1988) including from the New England states in the east to Oregon and California in the west. It is known from many provinces in Canada and may now have been found from people in most provinces. For eastern Canada there are published reports of human cases in at least Nova Scotia, Quebec and Ontario. Our knomdedge of the distribution is expanding rapidly as understanding of the disease in the medical profession, and surveillance for disease in Canada is heightened. In 1988, Lyme disease officially became a reportable disease in Ontario.

Suggestions that Lyme disease may be global in distribution was supported by the first descriptions of the disease in Australia in 1982.

There are many suggestions that Lyme disease is spreading geographically. These suggestions are substantiated by comparative surveys over time in some enzootic areas in New York, Connecticut and Wisconsin. However, for many areas it is not possible at this time to interpret whether new discoveries of Lyme disease represent a recent spread of the disease to these areas or represent more knowledge and more efficent survey techniques than have been available previously. I personally suspect that some of the apparent rapid "expansion" of the distribution of Lyme disease cannot be logically explained in an epizootologic context. This is despite the fact that birds are recognized as being capable of disseminating infected ticks which may be parasitic on them (Anderson et al. 1986).

Host Distibution:

The main vertebrate hosts of **B. burgdorferi** are **Peromyscus** spp. and white-tailed deer (Bosler et al. 1984, Levine et al. 1985). However, spirochetes have been isolated from additional mammals including **Microtus pennsylvanicus** (Anderson et al. 1986), raccoon (Anderson et al. 1983), coyotes (Burgess and Windberg 1989), black bears (Kazmierczak et al. 1988), black-tailed jack rabbits (Lane and Burgdorfer 1988, Burgess and Windberg 1989), and Columbian black-tailed deer (Lane and Brugdorhz 1988h

Antibodies to **B. burgdorferi** have been recovered from 0.3 to 50% of opossums, eastern chipmunks, raccoons, and grey squirrels in addition to white-footed mice and deer in Conneticut (Magnarelli et al. 1984). Kazmierczak et al. (1988) found antibody to **B. burgdorferi** in a wild timber wolf.

Domestic animals from which **B. burgdorferi** have been recovered include dogs (Magnarelli et al 1984), horses (Marcus et al. 1985), and cattle (Burgess et al. 1987).

Native North American ticks that have been implicated as hosts of **B. burgdorferi** include the main vector in the east, **Ixodes dammini** (Burgdorfer et al. 1982), **I. pacificus** (Burgdorfer et al. 1985), **Dermacentor variabilis** (Anderson and Magnarelli 1984), **Amblyomma americanum** (Schulze et al. 1984), and **Ixodes neotomae, Haemaphysalis leporispalustris**, **D. occidentalis**, and **D. parumapertus** (see Lane and Burgdorfer 1988)

Magnarelli and Anderson (1988) report preliminary results that may, with further research, implicate some tabanids and culicids as also being potential vectors of **B. burgdorferi**.

Ticks remain the primary vectors, within our current knowledge, but our understanding of both the vertebrate and invertebrate host distribution will continue to expand rapidly within the next 5 or more years.

Pathology:

In humans, early stages of the disease may be manifested by ECM in some but not all patients. That is the extent of the disease in some patients. However, ECM may be accompanied by headache, feven chills, malaise, fatigue and regional lyinphadenopathy.

Weeks to months later neurolgic complications (e.g. meningitis) and cardiac disorders (more rare) may be expressed. About 60% of patients develop arthritis within a few weeks to 2 years after onset of illness. The arthritis may become chronic with erosion of cartilage and bone (Steere et al. 1984).

It has been suggested that **B. burgdorferi** may be one cause of Bell's palsy (Schmutzhard and Stanek 1985) and multiple sclerosis (Kurtz 1986). In addition, infection with **B. burgdorferi** in pregnant women has been suggested as a possible cause in disproportionately high occurrences of adverse pregnancies (Markowitz et al. 1986).

Problems in pregnancies have ranged from rashes in the infants and premature births to blindness and intrauterine fetal death.

In dogs, clinical disease was absent from dogs inoculated with **B. burgdorferi** (Greene et al. 1988). One of 4 wolves inoculated with **B. burgdorferi** developed lyraphadenopathy but disease was otherwise absent clinically and at post mortem in these wolves (Kazmierczak et al. 1988).

Arthritis has been associated with infection in a Wisconsin pony (Burgess et al. 1986). Nine of 86 horses with antibodies to **B. burgdorferi** in Connecticut and New York developed limb or joint disorders that resulted in lameness (Magnarelh et al. 1988). Encephalitis has also been associated wiua **B. burgdorferi** in a horse (Burgess and Mattison 1987).

In cattle, both arthritis and systemic complications have been reported (Burgess et al. 1987).

Public Health Implications:

The implications are obvious based on the description of clinical disease given.

Transmission:

White-footed mice are the most competent reservoir host for **B. burgdorferi** known to date (Donahue et al. 1987). Spirochetes can be transmitted via the bites of **I. dammini** and other ticks as described above. Spirochetes can also be shed in urine of mice (Bosler and SchWze 1986). Burgess et al. (1986) demonstrated that transmission can occur by contact between mice. Oral infection between mice is another form of transmission (Burgess and Patrican 1987). Transovarial transmission is also known to occur within tick populations (Piesraoui et al. 1986, Lane and Burgdorfer 1987).

Transmission between areas is complicated by the potential for birds to disperse infected ticks over broad areas (Anderson et al. 1986).

Humans most frequently contract the disease during summer when the host-seeking behaviour of nymphal **I. dammini** is greatest (Piesman et al. 1987). It has been demonstrated that transmission of **B. burgdorferi** from infected ticks to rodents is less successful if the ticks are removed within 24 hrs, as compared to 48 and 74 hrs (Piesman et al. 19871

The diversity of known vectors and known methods of transmission of **B. burgdorferi** is pronounced. The possible involvement of other hematophagous anthropods as vectors (Anderson and Magnarelli 1988) serves to emphasize that much more is yet to be learned about transmission of **B. burgdorferi**. Of particular importance, is that the potential for deer to serve as a reservoir for infection to humans or other animals is questionable (Telford et al. 1988).

Diagnosis:

In their initial work identifying the spirochaete as the agent of Lyme disease, Burgdorfer et al. (1982) cultured spirochetes in a medium called Kelly's medium and used an indirect immunofluorescence assay (IGFA) method to identify specific antibodies from blood.

The reliability of EFA and enzyme-linked immunosorbent assay (ELISA) diagnostic techniques can be high, particularly during advanced and more complicated Lyme disease cases (Russell et al. 1984). Russell et al. (1984) found reduced reliability of the specific diagnosis of Lyme disease in patients with syphilis because of some cross reactivity between antibodies to **Treponema pallidum** and **Borrelia burgdorferi**.

Comparison of IFA and ELISA techniques for diagnosis of Lyme disease in humans, dogs, and white-footed mice indicated that positive responses were similar in 95% of 123 sera (Magnarelli et al. 1984).

There are diagnostic problems with early Lyme disease cases. Culturing was successful in isolating spirochetes in only 1 of 40 patients, and specific antibody response is delayed and not measurable during early phases (Shrestha et al. 1985). Detection of Lyme disease is often reliant on clinical findings during this early period. By 14 days after diagnosis of ECM, Magnarelli and Anderson (1986) reported specific antibodies present in slightly more than half their patients.

A problem of cross-reactivity with some other spirochaetal infections remains a problem when testing for Lyme disease. Magnarelli et al. (1987) found no cross reactivity between **B. burgdorferi** and **Leptospira interrogans**. However, they did find reactivity in Lyme disease patients with concurrent infections with tick-borne relapsing fever, louse-borne relapsing fever and syphillis. Serological results must obviously be interpreted with some caution.

Treatment:

In humans, early stages of Lyme disease have been treated much more effectively with penicillin or tetracycline than with erythromycin (Steere et al. 1983a). Steere et al. (1983b) found high doses of administered I.V. penicillin to be effective in controlling the disease with no after effects of patients who had exhibited neurologic abnormalities. However, with early Lyme disease, tetracychne appears to be more effective than does penicillin in controlling the disease and producing fewer subsequent complications (Steere et al. 1986). Steere et al. (1986) also recognized that penicillin given either IM or IV was effective in resolving the arthritis problem in 35-50% of patients. Dattwyler and Halperin (1987) found a treatment regime with tetracycline to be less effective than desired in treating early Lyme disease.

The results of this work demonstrate that use of some antibiotics at certain treatment levels may be more effective than other options at controlling a specific phase of Lyme disease in humans. Few published data are available to date on experimental treatment of Lyme disease in livestock.

Treatment against Lyme disease can also be directed at the vector tick population. There are a wide variety of effective acaricides available. Two which have been tested against **I. dammini** under field conditions and which have reduced the adult population by 97-100% are carbaryl and diazinon (Schulze et al. 1987).

Research Required:

There are many avenues for productive research useful to concerns in Saskatchewan. However, with the enormous research effort and funding being generated in other parts of the continent, I recommend that in Saskatchewan initial research efforts be restricted to delineating the extent of Lyme disease in the province. Valuable research dollars can be directed toward other diseases of local concern for which there are fewer results available from other sources. The most valuable epizootiological research at this time could be to establish the ability of cervid (or other large) hosts to serve as competent reservoirs of infection with **B. burgdorferi**.

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Group Discussion:

Can wildlife be readily monitored to see if they carry the disease?

Yes, wildlife can be monitored. The most common form of monitoring would be serological surveys for evidence of antibodies to **Borrella burgdorferl**. Wildlife can be seropositive without being a source of transmission of the bacterium to other hosts.

A more exact form of survey is to survey tissues of wildlife by attempting to culture the bacteria from them. In some areas, bacteria have been cultured from ticks in addition to some of their vertebrate hosts.

There remains no single diagnostic test for Lyme disease. The communicable Disease Center in Atlanta, Georgia uses a number of different criteria for a positive diagnosis. Criteria accepted by them include:

1) ECM (the skin rash) plus positive serology
2) ECM and culturing of the bacteria
3) positive serology + isolation of bacteria + one body system involved

Could you do an FA test?

With any test, it is the sensitivity and predictability of the test that matters rather than the type of test. CDC uses an ELISA test for serological evidence of infection. As indicated in the diagnosis section IFA and ELISA tests are both known to be effective.

Will Ivermectin take care of the worries of importing animals?

This is a difficult question to answer. Ivermectin can be effective against ticks that are in an actively feeding stage. However, even if the tick vectors are removed with use of Ivermectin, the imported animals could still be reservoirs for the bacteria. The disease could then spread locally when suitable biting arthropods fed on the imported reservoir animals. If further research were to demonstrate that cervids are poor reservoir hosts for the bacterium and if acaricides such as Ivermectin are effective, then and only then, will concerns about importing Lyme disease with imported cervids be of no worry.

Perhaps one problem that Samuel and coworkers had with the efficacy of Ivermectin as an acaricide on moose was that the winter tick was not in an active feeding stage. This is the case during much of its time on moose.

Can the Canadian government be convinced to licence acaricides not currently licensed?

This will probably happen over time and the products to be considered will almost certainly have been tested previously by the United States Dept. of Agriculture at their livestock pest experimental station in Kerrville, Texas. Contacting Dr. Roger Drummond at that station would bring one up-to-date on current developments with acaricides.

Should importation of game animals from enzootic areas for Lyme disease be considered?

]Definitily, not. There is serological evidence of a fairly high occurrence of **B. burgdorferi** in deer even from areas where ticks are almost unknown from deer and cases of Lyme disease in people are rare. This is the case from some recently unpublished data from Ontario. That being the case, one would be ill advised to import animals from areas where Lyme disease is enzootic.

*Can winter ticks be a problem as a vector of **B. burgdorferi**?*

Yes, but winter ticks are unlikely to be a problem in the wild. Winter ticks have been demonstrated under laboratory conditions to be capable of transmitting the disease. However, being a one host tick, under normal circumstances, it would be difficult to get transmission from one animal to another.

Transmission of the bacteria can occur transovarially, that is from an infected female tick through the eggs to the young ticks. The bacteria could cycle via winter ticks in the wild in this manner.

It is likely that winter ticks with a preference for feeding on moose may attack and potentially have an initial feeding on another species of wildlife before detaching and waiting for an opportunity to attach to a moose. It is not known that this happens in the wild, but it is likely that it occurs. Under these circumstances, a winter tick could feed on an infected small rodent or other carrier of the bacterium and then transmit the disease to moose.

What types of research would be most appropriate regarding Lyme Disease in Saskatchewan?

Monitoring sera of dogs, livestock, rabbits and hares and cervids (if blood is readily available) would be a suitable initial step in establishing the range of **B. burgdorferi** in Saskatchewan.

Q Fever

E. M. Addison
May, 1989

Disease:

Q fever, or abattoir fever, is a rickettsial disease, largely of livestock. It is also a zoonosis.

Agent:

The agent of Q fever is **Coxiella burnetti**. It was discovered in the late 1930's amongst abattoir workers in Australia (Derrick 1939) and about the same time from naturally infected ticks (**Dermacentor andersoni**) in the United States (Davis et al. 1938). The organism multiplies only within living cells and multiplies by binary fission. There are few strain differences that have been demonstrated amongst organisms from different parts of the world.

C. burnetti is uniquely different from most other rickettsiae by its resistance to physical and chemical agents. It has survived for at least 586 days in dried tick faeces (Philip 1948) and can survive in milk at 62°C for 30 min (Stoenner et al. 1959). The organism survives for at least 24h in 1% phenol and 1% formalin solutions (Ransom and Huebner 1951).

Geographic Distribution:

Q fever is essentially world-wide in distribution. It occurs mainly in countries in which cattle, sheep. and goats are raised in substantial numbers. It is considered to be widely distributed within the United States. The first identified case of Q fever in humans in Canada was in 1955. In a Quebec abattoir, 62 people were diagnosed with the disease in 1956. The disease was first known in domestic livestock in Canada huni Q fever antibodies in cattle from British Columbia in 1961. By 1962, serological evidence had demonstrated **C. burnetti** in cattle in Ontario and Quebec and in humans in Ontario (see McKiel et al. 1962). A study of small mammals and birds around Ontario farms with seropositive cattle implicated rats, a white-footed mouse and a barn swallow as possible reservoirs of infection (McKiel et al. 1962). I did not find specific information on the distribution of the organism in agricultural animals in Canada although I am sure that such information exists.

Host Distribution:

The range of hosts involved in the epizootiology of Q fever is extremely broad, extending from ticks to primates. Evaluation of the hosts has not provided any suggestion as to which pests are natural hosts and which might be alternate or refractory hosts. Studies of wildlife during epizootics of Q fever in California and Utah (Stoenner et al. 1959; Sidwell et al. 1964; Enlight et al. 1971a, 1971b) indicated that epizootics can be widespread amongst wildlife. Stoenner (1980) considers Q fever to occur in two cycles, one amongst wildlife and their ectoparasites, and the other amongst domestic animals and man. He also suggests that wildlife have been the origin of Q fever to domestic animals. Prevalence of the disease, as measured serologically, can be high in domestic herds. For example, in Ontario 17% of goat herds and 67% of cattle herds are estimated to contain seropositive individuals (Prescott and Lautenslager, unpublished).

Although there is serological evidence of infection amongst many North American wildlife and organisms have been cultured from many wildlife, Columbian black-tailed deer from California (see Enlight et al. 1971b) may be the only wild North American ungulate from which organisms have been cultured. Other ungulates that have been identified as seropositive include caribou (10%) in Alaska (Hopla 1975); a)ds deer (66%) and fallow deer (32%) in California (Riemann et al. 1979); Dall's sheep (80%) in Alaska (Zarnke 1983); caribou (0-67%), muskoxen (7%), Dall's sheep (0-33%), and mountain goats (15-33%) in Alaska (Zarnke 1986); and bison (17-33%) in Alaska (Zarn-ke 1988).

At the same time it is important to note that there are populations of wild ungulates that have been examined serologically for evidence of C. burnetti and have been found to be negative. Rice (1947) examined 185 elk and 36 bison from national parks in Alberta and found no evidence of infection. Similarly, Merrell and Wright (1978) found no seropositive samples in examination of 88 elk and 89 mule deer from central and northern Utah.

Many haematophagous arthropods are known to be naturally infected with **C. burnetti**. These include a wide variety of ixodid and argasid ticks, mites, fleas, lice, and tabanids (Krinsky 1976, Stoenner 1980).

Pathology:

The disease in livestock is sufficiently mild for some to question its designation as a disease in livestock. Clinical signs are generally absent (Bell 1981). Although large numbers of organisms can be isolated from fetuses of infected cattle and sheep and pregnant cattle inoculated with large numbers of organisms may abort, cattle are not known to abort because of infections under normal conditions. The number of organisms used to induce abortions under experimental conditions are in excess of the number experienced by catttle under natural conditions (see Stoenner 1980). Similarly, investigators had not recognized signs of clinical disease in sheep and goats that can be directly attributable to infection with **C. burnetti**, until 1976 when 20 of 33 dairy goats in one herd aborted and the causative organism in the fetuses was **C. burnetti** (Stoenner 1980). It may be that other cases with clinical pathology in domestic livestock have occurred since that time. If so, it emphasizes that although usually asymptomatic, Q fever may cause serious problems.

I know of no experimental infections with **C. burnetti** and follow up studies of pathology in wild ungulate species. In addition, almost nothing is known about naturally acquired Q fever in wild ungulates (Bell 1981).

Public Health Implications:

In humans, on the other hand, the disease can be serious and the pathology is well understood. The incubation period is usually 18-20 days but may be anywhere from 2 to 4 weeks (Stoenner 1980). The disease most commonly occurs in people over the age of 40 and seldom occurs in children under 10 year of age. Symptoms in humans can be varied but most commonly include fever, malaise, chills, general weakness, headaches, muscle pain, anorexia, sweating, coughs and nausea (see Stoenner 198O). Patients may be found with respiratory complications and less frequently with hepatomegaly, splenomegaly, irregular heart rates, and other conditions. In 1979, one fatality and 3 other cases of Q fever were associated with use of sheep fetuses in a U.S. medical school (Stoenner 1980).

Transmission:

Modes of transmission in the wild are unknown. However, the frequent occurrence of organisms in hematophagous arthropods and successful experimental transmission using biting arthropods suggesh that anthropods may be involved in the wild. The rickettsiae are eliminated from the bodies of infected animals in placentas, amniotic fluid, and milk. Because of the high concentration of organisms in placentas, they could be the source of infection to scavengers and other carnivores consuming placentas not consumed by the female bearing young.

Rickettsias have frequently been isolated from air in buildings holding infected livestock. These and other observations suggest that the most likely route of transmission amongst livestock is aerosol transmission. Man most likely also contracts the rickettsiae by inhaling organisms. Humans have been demonstrated to seroconvert after consuming infected milk but did not acquire any noticeable disease when coming into contact with the rickettsiae in this manner (Benson et al. 1963).

Urine, saliva, and phlegm of Q fever patients can be infective. However, there is little to no evidence that transmission occurs amongst humans (Stoenner 1980).

Diagnosis:

Sera in livestock and humans are tested for infection using similar techniques. A wide variety of available serological tests are available. Results from one type of test are not necessarily transposable to another test. All the serological tests require identification of an increasing number of antibodies during the infection. Bell (1980) indicates that selection of a particular test may depend on preference and prior experience. I recommend contacting an experienced person before selecting from the tests listed below. Tests include complement fixation, capillary agglutination, standard rickettsial agglutination, microscopic slide agglutination, agglutination resuspension, radioisotope precipitation test (RIP), opsonin test, serum neutralization, serum protection, direct and indirect fluorescent antibody, passive cutaneous anaphylaxis, and enzyme-lifted immunoassay.

Treatment:

There are no prescribed techniques for treating infections in wildlife or domestic animals, perhaps because of the absence of known serious disease directly attributable to **C. burnetti**.

In man, methods of treatment attempted have not been universally successful. Chloramphenicol and several tetracycline-group antibiotics are considered to stop development of disease but may not be kill the rickettsiae. Trimethoprim-sulfamethoxazole and rifampin are also used. Our inability to successfully and predictably treat cases of Q fever in humans is perhaps one reason that **Coxiella** has been an agent considered for germ warfare.

Research Required:

Because Q fever is often asymptomatic in livestock and there is no evidence of clinical pathology in infected wildlife, research on Q fever should not be considered as high a priority for game ranching concerns as would research on many other diseases more pathogenic to livestock and wild ungulates. NOTE THAT I HAVE NOT RECONMNDED THAT Q FEVER SHOULD NOT BE OF CONCERN IN GAME RANCHING OPERATIONS! In fact research on Q fever is important because we know almost nothing of its pathogenicity in wild ungulates and the pathology in domestics is seemingly unpredictable enough to warrant much further investigation.. The highest research priority is to establish, through experimental infections, the pathogenicity of the disease in various wild ungulates.

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Group Discussion:

Are cases in humans related to transmission by ticks or contact with an infected animal?

Almost all human cases are associated with contact with an infected animal. Infections are often acquired when handling infected carcasses on farms or in meat packing plants. Of greatest threat of infection are birth fluids and tissues from which large numbers of organisms can be isolated. However, human infections can even be established by inhalation of the organism.

Are there problems in diagnosis of Q fever?

Once antibodies to **C. burnetti** are present, diagnoses can be based on results of a wide variety of serological tests. However, results of one type of test should not be considered the same as results of some other tests. Different tests have differing levels of sensitivity.

Q fever in humans may be difficult to diagnose because, in acute infections, symptoms can be consistent with those of brucellosis, mononucleosis, infectious hepatitis, viral pneumonia, and influenza.

Is there an association between livestock and the likelihood of contacting Q-fever?

Yes. The disease in humans definitely occurs most frequently in areas where cattle, sheep, and goats are abundant. This is a very good reason for not mixing game farming with goat or sheep farming.

Bluetongue

Alan Kocan

Name of Disease:

Hemorrhagic Disease

Species Affected:

White-tailed deer

Type of Disease:

Viral

Scientific and Common Names of Disease:

Bluetongue and Epizootic hemorrhagic disease.

Known Geographic Distribution:

Worldwide with 5 known BTV serotypes and 2 EHDV types known in North America. Appear to be restricted to lower latitudes in North America.

Clinical signs:

Clinical signs are influenced by the specific serotype involved and the virulance of the virus incountered. Deer showing signs of BT infection generally experience a progressive episode beginning with depression, fever, and inactivity. Most animals progress to the point of lameness although mortalities may occur prior to this point and may reach as high as 50%. Mortalities due to virulent strains may occur as soon as 72 hours after exposure. Infection with EHDV generally follows the same clinical course, depending on the virulence and strain of virus involved.

Major Gross Pathologic Changes:

One of three distinct forms may be seen: 1) The paracute or rapid form generally is characterized by severe edema of the neck, head, tongue, and lungs, 2) the acute or "classical" form is characteized by edema coupled with hemorrhages and/or congestion of the heart, rumen, and intestines; areas of necrossis or ulceration may be present on the dental pads, tongue, palate, rumen, and abomasum, 3) the chronic form is characterized by growth interruptions of the hooves or sloughing of the hoof walls along with ulcerations on the rumen mucossa.

Microscopic Changes:

Microscopic lesions are seen as edema and hemorrhages and/or ulcerations of various organs. Dental pad, tongue, palate, rumen and omasum are generally involved but hemorrhages, necrosis or ulcerations may occur in any organ.

Method of Diagnosis:

Viral isolation and typing is necessary for confirmation of BTV or EHDV. Routine isolation attempts involve inoculation of embryonated chicken eggs followed by inoculation of homogenized chick embryos onto 1-day-old preformed African green monkey kidney (Vero-Maru) cells. Serology (microager-gel precipitin test)) is useful to detect viral activity in a herd but should not be used alone as a means of determination of infection. Presence of antibody does not mean that virus is present and only indicates exposure. Serology cannot determine the serotype involved. Gross lesions may be used as a means of establishing a tentative diagnosis but similar lesions are produced by a large number of viral agents.

Method of Treatment and Treatment Effectiveness:

No treatment is presently available for free-ranging species. Previous exposure appears to provide protection but only for the specific serotype involved. Increasing evidence suggests that endemic stability may occur in some areas due to continuous endemic transmission. In these instances, prevalence is near 100% and transmission occurs on a yearly basis. Stability appears to be related to a single serotype and introduction of other serotypes or strains will probably result in clinical illness.

Other Species Affected By The Same Organisms:

A wide variety of wild and domestic ruminants are susceptable to infection with both viruses. The course of infection varies from being asymptomatic to acute. Clinical signs are similar to those described for deer.

Clinical Signs in Other Species Affected:

Both EHD and BT are known to infect a variety of wild and domestic ruminants. In domestic sheep and white-tailed deer, clinical signs develop rapidly, with the incubation and viremic period being short, therefore these animals are probably short term reservoirs, especially for BTV. Cattle, goats, and elk display a subacute or inapparent respnse to BTV and are generally considered long-term carriers. other wild ruminants known to be susceptable to BTV include antelope, bighorn sheep, mule deer, and exotic species of bovids and cervids. Recent studies have shown that enzootic stability to certain serotypes may occur in certain areas for both viruses, thus leaving white-tailed deer, and perhaps other species, as inapparent carriers. Malformed and/or aborted fetuses have been reported in domestic sheep and cattle as well as some exotic ruminants with acute BTV infections.

Prevelance of the Disease in Wild and Domestic Species:

Serologic prevalence can range as high as 100% in free-ranging herds and appears to be limited only by the presence and abundance of the vector. In North America, the biting midges of the genus **Culicoides**, especially **C. varipennis**, are the major vector. Disease generally occurs during the vector season, late summer to early fall, ending with the first killing frost. Transmission by contact has not been documented and as a result, vector activity and prevalence are important in the epizoodiology of the disease. Animals with the chronic form of the disease may be seen in winter, after the vector season. Detectable virus is generally not present in these chronically infected animals.

Importance to Domestic Species:

Both BTV and EHDV are capable of infecting a wide range of domestic ruminants. Clinical disease is common with infections with either virus. Regulatory constraints presently exist for animals and seman found to be infected with these viruses.

Public Health Implications:

None known.

Research needed:

Determination of distribution within geographic areas and documentation of specific serotypes. Serologic studies provide vary little insight into the true epidemiology of these viruses and viral isolation and typing are needed to better understand the total picture.

Group Discussion:

How reliable are serologic tests in determining infection?

Serology can only detect exposure. In many cases, seroreactive animals are not carrying virus and in some cases, seronegative animals do cary virus. At best, serologic tests are a good screening tool especially in determining viral activity in an area. However, present regulations restrict importation of animals into Canada that are seropositive. Keep in mind that isolation of this virus is not easy and most laboratories are not equipped to handle isolations or experienced enough to deal with field isolations.

Does the epidemiology of bluetongue in wildlife differ in different locations throughout North America?

Apparently, there is considerable difference in the epidemiology of the disease between northern and southern portions of North America. In southern regions where vector activity is apparently more constant from year to year, a high prevalence of seroreactivity in most ruminant herds is common. As you go further north, prevalence is much lower. These apparent differences are probably related to vector activity and consistancy in transmission from year to year. In some areas in the US, such as Wyoming, increased bluetongue activity can usually be detected early by noted mortalities in susceptable wildlife species such as antelope and deer. In southern regions, such as Oklahoma, seroreactivity in most deer herds is close to 100% of adult animals. The difference is probably related to more consistant yearly activity of vectors in the southern and sporatic vector activity in the north.

How long can animals carry the virus?

The ability of animals to carry virus appears to vary with the animal involved. Cattle and probably elk are considered long term carriers. Additionally, there appears to be little mortality associated with infections in these species, with ony occasional congenital and neonatal abnormalities and deaths. Other species such as sheep and perhaps deer may be more susceptible to clinical disease and appear to be short term carriers. Keep in mind that all that is needed for transmission of BTV is the organism, the proper vector and susceptible animals. In northern regions, all three components are present but perhaps not on as regular a basis as occurs in the southern areas. This might explain why outbreaks and apparent transmission is more sporadic in northern areas. Another possible explanation might be related to the fact that long term reservoirs such as cattle are not routinely kept alive for more than a few years. As a result, the source of infection may be reduced because of a lack of long term reservoirs.

Is it safe to move animals during an outbreak of BT?

If you want to be safe, and insure that the movement of the virus is restricted, don't move the animals. Timing is an important factor in the spread of this virus. If you wait until after the vector season is over, (September) you will reduce the chances of transmission. Be aware that some species may be long term carriers and the problem may reappear at the next vector season. Also be aware that many ruminants are capable of being infected and carrying the virus and restriction on one host may not contain the spread of the organism. Game ranchers should be especially careful in movement of animals since some ranched species are extremely susceptible to disease and introduction into an area may have devastating effects.

What are the most pressing reseach needs related to bluetongue in wildlife?

A serologic test with greater sensitivity and specificity is needed. We also are in need of experimental studies that will determine susceptibility of different wildlife species to differing serotypes of BTV. Information related to length of viremic period. Cross protection between serotypes, effects of repeated exposure and modifications in the virus when transmitted from wildlife to domestic animals and vise versa are needed.

Elaeophora schneideri

Alan Kocan

Name of disease:

Elaeophorosis

Species Affected:

White-tailed deer

Type of disease:

Parasitic

Scientific and Common Name of the Disease:

Elaeophora schneideri,Elaeophorosis, clear-eyed blindness, arterial worm.

Known Geographic Distribution:

Originally considered a parasite of western states, it has now been reported to occur in several southern states and its range may be broader than previously thought. Reports exist from New Mexico, Colorado, California, Utah, Wyoming, Montana, Florida, Georgia, Oklahoma, Texas and western Canada.

Clinical Signs:

Signs vary depending on the host infected. Mule deer and black-tailed deer are considered normal definitive hosts and show no clinical effects from infection. Abnormal hosts such as elk, moose, white-tailed deer, domestic sheep and goats show signs ranging from dermatitis to death. Clinical signs in young animals are generally more severe than in adults.

Major Gross Pathologic Changes:

In many abnormal hosts, gross pathologic changes include blindness, cropping of the ears, necrosis of the muzzle, antler deformaties and damage to the central nervous system. Reports of mandibular abnormalities in white-tailed deer have also been noted.

Microscopic changes:

Microscopic lesions in abnormal hosts that survive the initial infection include post-necrotic cavitation of the cerebral hemisphere resulting from lack of blood supply to this region of the brain. Parasites can be seen in leptomeningeal or cerebral arteries with blood clots and thrombi occuring distal to this point. Necrosis due to restricted blood flow is common in many tissues due to movement of the parasite. One preferred site is the renal artery and its terminal branches. Parasites have also been found in coronary arteries, mesenteric arteries, digital arteries, iliac and femeral arteries.

Method of Diagnosis:

In both normal and abnormal definitive hosts, adult worms inhabit the common carotid arteries. Microfilaria of this nematode can be found lodged in the arterioles and capillaries of the skin associated with the forehead and facal region. The microfilaria are large (275 x 18 microns) which does not allow them to be passively carried throughout the circulatory system. In the mule deer host, diagnosis can be made by scraping the skin of the forehead and identifying the microcfilaria by microscopic examination. Adult worms can be recovered from the corotid arteries. Adult female worms are 60-120 mm long x 0.6 - 0.9 mm thick. Adult males are 55-

85 mm x 0.4-0.7 mm thick. Both sexes have three prominent conical protruberances on the distal extremity. The most important feature used in identification of adult worms is the morphology of the left spicule of the male which has a distinct eyelet-like aperture at approximately the middle of its length. Determination of infection in the abnormal host requires removal of the brain and stripping the meninges. The meninges are then digested in pepsin-hydrochloric acid (10%) and examination for the presence of the developing parasite. In abnormal hosts that have survived the initial phase of the infection, diagnosis can be made by examining the corotid arteries, the aorta, and left ventrical of the heart for the nematode parasites.

Other Species Affected by the Same Parasite:

Susceptable animals include domestic goats and sheep, elk, moose, and probably wild sheep.

Clinical Signs in Additional Species:

Clinical signs in other species (abnormal hosts) include dermatitis asociated with the head and poll region, blindness, cropping of the ears and/or muzzle, antler deformation, and central nervous system damage. Most changes are related to aschemic necrosis caused by the developing worms in the abnormal host.

Prevaleance of the Disease in Wild and Domestic species:

Prevalence in mule deer in endemic areas may range from 10% to as high as 100%. In New Mexico, west of Raton, prevalence in mule deer herds reaches 80-90% while further south, mule deer that inhabit areas over 5,000 feet in elivation generally show 100% prevalence of infection. In Colorado, prevalence generally ranges from 10-25% while prevalence in California has been reported to range from 4% to 44%. The parasite has been reported for numerous other western states in the United States including Utah, Wyoming, Montana as well as western Canada. Reports also exist from Florida, Georgia, Oklahoma and south-central Texas. Overall prevalence will vary depending on the mule deer population and the abundance of the vector (horseflies). Prevalence data for white-tailed deer is incomplete.

Importance to Domestic Species:

Domestic sheep and goats are susceptable to infection with this nematode parasite. Experimental infections in both domestic sheep and goats have shown that blindness and severe brain damage could occur, beginning 7-10 days after exposure. Animals that survived beyond this period developed a characteristic allergic dermatitis. The dermatitis, termed 'sorehead" usually begins in the poll region and spreads over the face. This condition is characterized by intense itching, with periods of intermittent and incomplete healing which may persist for years.

Public Health Implications:

None known.

Research needed:

Determination of exact geographic distribution east of the Rocky Mountains. Clinical trials with antihelminthic products that are known to have larvacidal effects are warranted.

Group Discussion:

Is elaeophorosis a potential problem for animals in Canada?

The potential is certainly there for this being a problem. As in any disease, the three most important ingredients that are necessary for spread are a source of infection, a means of infection, in this case the horsefly vector, and susceptible hosts. You have the necessary vector in horseflies and a suitable host in mule deer. Transmission can occur if you have a source of infection. This can come from infected mule deer, from domestic sheep and goats and possibly other abnormal hosts such as white-tailed deer or elk.

There is a great deal of similarity between the possible introduction of this organism and meningeal worm in Canada. At present you do not appear to have the parasite in Saskatchewan or Alberta. It is possible that all that is necessary for establishing the infection is the introduction of the parasite. This can be accomplished by movement of game animals from the U.S. will surely increase the chances of its introduction.

Are the diagnostic tests specific enough to minimize the possibility of introduction of the parasite?

In general, yes. The microfilaria is both morphologically and anatomically distinct. The location from which it is isolated is also unique. If you are looking for it, you should have little problem in diagnosing it. Care must be taken, however, to test any susceptible animals coming from endemic areas. Two tests may be necessary to insure that animals are not infected. One test should be conducted in the winter and a second beyond the prepatient period.

Lungworm

Alan Kocan

Name of the Disease:

Lungworm

Species Affected:

White-tailed deer

Type of Disease:

Parasitic

Scientific Name and Common Name:

Dictyocaulus viviparis, lungworm, parasitic bronchitis, "Husk", "Hoose".

Known Geographic Distribution:

Lungworm infections in white-tailed deer have been reported from most of its range. The parasite is known to have a world wide distribution.

Clinical signs:

Most adult deer do not show clinical signs of infection with this nematode parasite. Deer with heavy infections may show weight loss and respiratory distress. Young animals and animals in poor nutritional conditions are most subject to clinical disease. Mortalities in these animals are most common in winter and early spring.

Major Gross Pathologic Changes:

Gross pathologic changes are restricted to the respiratory tract. Blockage of respiratory bronchioles with exodate and collapse of alveoli with an accompioning emphysema are common. A common occurance is a patchy pneumonia appearing as firm dark red to gray areas in the lungs. Fibrin on the surface of the lung and individual fibrotic lung lobules are also common. Excessive froth and mucus may also be present in the airways. Secondary bacterial pneumonia may also result.

Microscopic Changes:

Bronchial blockage and consolidation with emphysema are common. Eggs and larvae in the bronchioles and alveoli which lead to consolidation of lung lobules are common in patent infections.

Method of Diagnosis:

Diagnosis in the live animal can be accomplished by microscopic examination of feces containig characteristic first stage larvae. These larvae are 0.3 - 0.36 mm long and have no anterior knob, but the intestinal cells contain numerous brownish granules. Diagnosis at necropsy can be accomplished by visualization of large (1 1/2 inches in length), white nematodes in the trachea, bronchi, or smaller air passages of the lungs. In general, it takes 3-4 weeks after ingestion of infective larvae for adult worms to be present in the lungs.

Method of Treatment:

No treatment is practical for free ranging wildlife. Captive or farmed animals can be treated with Ivermectin at 100 micrograms/kg body weight with good results. A vaccine using irradiated larvae has been commercially avalable for domestic animals.

Other Species Affected by the Same Organism:

Most free ranging ruminants are susceptible to infection. Deer occupying range with infected cattle or other ruminants or deer in over populated conditions are most subject to disease. Reports occur for infections in wildlife species including mule deer, elk, moose, and domestic cattle, sheep and goats.

Clinical Signs in Other Species:

Clinical signs are similar for all ruminants

Prevelance of the Disease in Wild and Domestic Animals:

Prevelance can range from a few infected individuals to 100%.

Importance to Domestic Species:

Lungworm infection due to **D. viviparus** can be of considerable importance in all ruminants. Problems ranging from poor performance to death have been reported for cattle, goats and sheep. Young animals (under 1 year of age) and animals with other parasitic infections and/or those in poor nutritional condition are most subject to clinical disease. Most clinical cases are seen in stressed animals or animals with concurrent problems. most cases in young animals are seen during winter or early spring.

Public Health Implications:

None known.

Research Needed:

Documentation of endemic free-ranging populations.

Nematodes

Alan Kocan

Name of Disease:

Abomasal parasites of deer; large and small stomach worms of deer.

Species Affected:

White-tailed deer

Type of Disease:

Parasitic

Scientific and Common Name of Disease:

Nematode parasites found in the abomasum of white-tailed deer are refered to as stomach worms. There are 4 genera and 9 species of nematodes that are commonly encountered; **Hemonchus contortus**, **Apteragia odocoilei**, **A. pursglovei**, **Ostertagia mossi**, **0. dikmansi**, **0. dikmansi**, **Trichostrongylus axei.**, **T. askivali**, and **T. dosteri**.

Known Geographic Distribution:

Throughout North America.

Clinical signs:

Clinical signs can range from being inaparrent to death. Most common signs encountered are weight loss, emaciation, adema, and possibly reduced reproductive potential.

Major Gross Pathologic Changes:

Gross changes include emaciation, anemia, adema, and hemorrhage of the abomasum.

Microscopic Changes:

Microscopic changes are generally related to attachment of the parasites to the abomasal wall. Additional changes are seen relative to anemia and adema.

Method of Diagnosis:

Diagnosis of parasitic infectins due to nematode parasites can be accomplished by routine examination of feces that contain the eggs of these parasites. Microscopic examination can assist in determining the presence of nematode parasites, but identification of specific species and determinations of the level of infection require the inumeration of the adult worms in the abomasum and identification of individuals. The following key is provided as a means of identifying paraites of the abomasum of white-tailed deer from North America. The key is based on characteristics of adult male parasites only.

1) a) Easily seen with the naked eye (10-15 mm). Asymetrical bursa with spicules of equal length with barbs near the tip.....**Haemonchus contortus**.

b) Medium size (6-8 mm), spicules equal in length, gubernaculum present or absent.....2.

c) Small size (males less than 4 mm). Spicules unequal in length, gubernaculum present.....5.

2) a) Gubernaculum not present, spicules massive and trifucate, a helmet like cap present at the distal end of the branch.....3.

b) Gubernaculum present, spicules long and slender.....4.

3) a) Spicules with semicircular indentation on inside edge at distal end, lightly sclerotized.....**Apteragia pursglovei**.

b) Spicules without semicircular indentation on inside edge of distal end, heavily sclerotized (dark brown in color).....**Apteragia ococoilei (Skriabinagia odocoilei**)

4) a) Gubernaculum paddle-shaped, spicule tips rounded.....**Ostertagia mossi**

b) Gubernaculum rod-shaped, inside edge of spicules may appear granulated or foamy, spicule tips flattened in lateral view.....**Ostertagia dikmansi**

c) Gubernaculum paddle-shaped, spicules tip club shaped, massive.....**Ostertagia ostertagi**

5) a) Gubernaculum slender, lancer-shaped, about 1/2 length of spicules**Trichostronqylus axei**

b) Gubernaculum elongated, diamond-shaped, less than 1/2 the length of the spicules. 1 wide and 1 narrow spicule.....**Trichostrongylus askivali**

c) Spicules large and heavily sclerotized, gubernaculum massive, heavily sclerotized and tapering sharply, in lateral view.....**Trichostronqylus dosteri**

The procedure for conducting an abomasal parasite count (APC) is as follows:

Open and empty the contents of the abomasum into a large pan or pail. Scrape the mucosa lightly and wash the scrapings into the pan with water. Pour the contents of the pan into a 1 galon jug and preserve with 10% formalin. The contents of the jug is then poured slowly through a 100 mesh U.S. standard sieve and washed well with a fine spray of water. The material remaining in the sieve is then transfered to an Erlenmyer flask and water is added to bring the volume to 1000 ml. The flask is shaken to mix the contents and a 50 ml aliquot is poured from the mixture. The aliquot may then be diluted with water in a large beaker to facilitate examination. Portions of the diluted aliquot are then poured into a gridded petri dish and examined under magnification until the entire sample is examined. All worms are counted and removed so that they can later be identified. Worms are stored in 5 % formalin. The APC is calculated by multiplying the total number of worms found in the 50 ml aliquot by 20. (50 x 20 = lOOOml). If total numbers are below 10, the procedure is conducted 3 times and a average number is recorded.

The following is provided as an aid in interpreting results. Be aware that the guidelines were developed for white-tailed deer from the southeastern U.S. Additionally, the values are intnded to be useful only when average values from atleast 5 deer are used and when deer are taken during July and August.

Average APC	Deer Population Status
< 500	Below carrying capacity. Indicative of an expanding herd.
500 - 1,500	Within carrying capacity. Herd should not be allowed to expand beyond this level.
> 1,500	Herd exceeds carrying capacity. Herd reduction is needed. Mortality risk is increased.

It should be remembered that the presence of a few parasites is not of any consequence. When the numbers increase beyond normal levels, the mortality and morbitity risk increases. Although definitive studies for wildlife species are lacking, the presence of over 1,000 worms of any species should be considered indicative of disease. When **Hemonchus contortus** is involved, an excess of 30 worms per pound of body weight can be considered indicative of disease.

Method of Treatment:

Treatment of free-ranging animals is not practical. Ivermectin, given at a dosage of 100 micrograms per kg of body weight is sufficient for eliminating all abomasal nematodes in captive deer.

Other Species Affected by the Same Organisms:

The abomasal nematodes described here are infective for most species of domestic and wild ruminants. Reports exist for deer, elk, moose, antelope, bison, wild sheep as well as for domestic cattle, sheep and goats.

Clinical Signs for Additional Species Affected:

Clinical signs in all ruminants are similar as those described for deer.

Prevalence of the Disease in Wild and Domestic Species:

Prevelance depends on the number of animals infected and the variety of susceptible hosts occupying common range. Adult animals appear to have some degree of immunity but remain a source of infecton. Most reports of mortalities due to stomach worms in wildlife are in animals less than 1 year of age from overpopulated or semi-captive situations.

Importance to Domestic Species:

Since most wild and domestic ruminants are susceptible to infection by the stomach worms described here, both inaparrent and acute disease are possible. All ruminants occupying common range should be considered as a common pool for these parasites. Control of infections depends on antihelminthic treatment and controlling herd sizes and potential transmission.

Public Health Implications:

None known.

Research Needed:

Improved methods of treatment of free-ranging animals and critical studies designed to determine risk factors and clinical levels of infection.

Theileria cervi

Alan Kocan

Name of disease:

Theileriosis

Species Affected:

White-tailed deer

Type of disease:

Parasitic

Scientific and Common Name of the Disease:

Theileria cervi.

Known Geographic Distribution:

Theileria cervi is found in white-tailed deer throughout most of the southeastern U.S. The limiting factor in its distribution appears to be the distribution of its tick vector, the Lone Star tick, **Amblyomma americanum**.

Clinical Signs:

Observable clinical signs in non-splenectomized white-tailed deer have not been reported except for indications of mild anemia in fawns. Splenectomized, immuno-compromized or stressed animals (especially fawns) may show more notable anemias.

Major Gross Pathologic Changes:

Gross pathologic changes have not been reported. Mild lymph node swelling may occur shortly after infection.

Microscopic Changes:

Microscopic changes have not been reported.

Method of Diagnosis:

Diagnosis is made by microscopic examination of stained thin-blood films. Organisms (piroplasms) are pleomorphic but are typically pyriform to dumb-bell shaped with the most common form being paper-clip in appearance.

Method of Treatment:

None available for deer.

Other Species Affected by the Same Organism:

White-tailed deer appear to be the only species affected. Recent reports indicate that fallow deer (**Dama dama**) are not susceptable to the strains of parsite from the southern U.S. Susceptability studies in other species have not been conducted.

Clinical Signs in Other Species:

No reports exist related to susceptability of other species.

Prevalence of the Disease in Wild and Domestic Species:

Prevalence in endemic areas often reaches 100%. Infection appears to be common shortly after birth with young fawns showing parasitemias greater than 1%. In southern regions of the U.S., the parasite appears to over-winter in adult ticks. Infections are also known to be transstadially transmitted between nymphal and adult stages. Transmission in nature probably results from infecton by adult ticks that over-wintered. These ticks became infected the previous year by feeding on infected deer as nymphs. The over-wintering adults then feed on fawns, perpetuating the infection. Trans-ovarial transmission is not known for this group of parasites.

Importance to Domestic Species:

No domestic species are known to be susceptable to infection. However, critical studies have not been conducted.

Public Health Implications:

None Known.

Research Needed:

Documentation of distribution and susceptability of additional species.

Babesia odocoioei

Alan Kocan

Name of Disease:

Cervid babesiosis

Species Affected:

White-tailed deer

Type of disease:

Parasitic

Scientific Name and Common Name:

Babesia odocollel

Known Geographic Distribution:

The known distribution includes parts of Texas and Oklahoma and a single reporting from the state of Virginia. The actual distribution is probably much greater than is now known. Although published documentation is not available, preliminary studies have indicated that the natural vector is the ixodid tick, **Ixodes scipularis**. As such, the probable distribution of the organism may coinside with the distribution of this tick.

Clinical Signs:

Clinical signs in non-splenectomized deer appear to be minimal. In splenectomized and immuno-compromized deer, severe, acute anemia develops often ending in acute death. Possible problems can arise from neive stressed animals that are brought to endemic areas and exposed to infection.

Major Gross Pathologic Changes:

Gross pathologic changes are related to anemia.

Microscopic Changes:

Microscopic changes are related to anemia.

Method of Diagnosis:

Diagnosis is dependent on microscopic examination of stained thin-blood films and identification of the organism. The organism is ovoid to pyriform and occurs singally. Infection rates are generally below 1%. Because of the low level parasitemias, whole-blood cultures and sub-inoculaton into susceptable animals may be necessary. A serologic test (IFA) is available.

Method of Treatment:

None available.

Other Species Affected:

No other species are known to be susceptable. Definitive studies have not been conducted on other wildlife species. Domestic cattle appear to be refractive to infection.

Clinical signs:

None reported.

Importance to Domestic Species:

None reported but definitive studies have not been conducted.

Public Health Implications:

None known.

Research Needed:

Documentation of geographic range and susceptibility of other species.

Pseudorabies

Alan Kocan

Species Affected:

Swine

Name of Disease:

Pseudorabies, Aujesky's disease.

Type of Disease:

Viral (herpesvirus).

Scientific and Common Name of Disease:

Pseutorabies, Auiesky's disease, "mad itch".

Known Geographic Distribution:

Throughout North America.

Clinical Signs:

Clinical signs of pseudorabies can vary from being inapparent to fatal. Clinical signs vary considerably with the age of the pig. Young pigs, less than 4 weeks of age, often show high death losses. Fever, vomiting, tremors, incoordination and convulsions are common in these animals. Infected adult swine show signs of fever and upper respiratory inflammation but rarely die. Abortion and mummified fetuses have been reported from infections in pregnant sows. Transmission of the virus among swine is by nasal and oral secretions. Animal to animal contact is common but transmission of the virus can also occur via contaminated aerosols, food, water or objects. Infected carcasses may also be an important means of transmission and contamination.

Major Gross Pathologic Changes:

Gross pathologic changes in swine include inflammation of the meninges and inflammation of the nasal and throat mucosa. Small hemorrhages are generally present in the lymph nodes and kidneys.

Microscopic Changes:

Prinary microscopic lesions in hogs are degenerative and inflammatory changes in nervous tissue of the brain.

Method of Diagnosis:

A tentative diagnosis in hogs can be made based on clinical signs, and gross and microscopic pathologic changes. A definitive diagnosis can only be made by demonstrating the virus in brain tissue or tonsils by the florescent antibody test. The pseudorabies virus can also be grown in tissue culture that has been inoculated with material from the infected animal. Brain, nervous tissue, tonsil,lymph node and frozen serum are the prefered material for virus isolations. Serologic tests are often used to detect antibody in latent carrier swine.

Method of Treatment:

No known treatment. A new vaccine is comming onto the market (Upjohn Co) for use in domestic hogs. No trials are available for wild species.

Other Species Affected by the Same Organism:

A large number of wild and domestic animals are known to be susceptible to infection by the pseudorabies virus. Affected animals include cattle, sheep, horses, dogs, cats, foxes, raccoons, skunks, rats mice and perhaps birds. Mortalities in non-swine hosts showing clinical signs often reach 100%. Because of this high mortality rate, non-swine hosts are considered accidental dead-end hosts for this virus.

Clinical Signs in Other Wild and Domestic Species:

In all naturally or experimentally infected non-swine hosts, the virus produces an acute infection of the central nervous system resulting in loss of appitite, depression, staggering, spasms, convulsions and death. Self mutilation resulting from persistant scratching, biting or rubbing often occurs in non-swine hosts. The term "mad itch" is commonly used to describe this behavior.

Prevalence of the Disease in Wild and Domestic Animals:

Routine serologic testing of domestic swine has reduced the prevalence of the disease in North America. Many wild swine populatins contain seropositive animals that may be latent carriers. Spread from these animals to domestic swine as well as to other wildlife is likely. Definitive studies dealing with the impact of wild swine and this disease are lacking. Movement of wild swine without testing is not advisable

Importance to Domestic Species:

Psuedorabies is a major disease problem for the domestic hog industry. The role of wildlife species, other than swine, is probably not significant in the spread of the disease since clinical signs and mortalities are high. The importance of wild swine in the spread and maintenance of this disease appears considerable.

Public Health Implications:

Humans appear to be refractive to infection with this virus.

Research needed:

Documentation of infected wild swine populations and edipemiologic evaluations of their role in maintenance and spread of the virus.

Group Discussion:

What is the present status of the pseudorabies erradication program in the US?

A major effort is underway to erradicate pseudorabies from the US. A major obstacle appears to be feral swine, especially in the southern states. Identification and eliminations of these feral herds is a major problem because most are free-ranging and capture and removal is difficult. The spread and maintenance of pseudorabies inthese feral populations is a major concern.

What is the status of the problem in Canada?

In Canada, pseudorabies is a reportable disease and when it is reported, the source is eradicated. In general, there are no feral swine in Canada so the reservoir problem is not present. Interest in the importation of wild boar is closely watched and pseudorabies is tested for. With the apparent increased interest in importations of wild boar, special attention must be paid to this problem since the swine industry in Canada is of considerable economic importance.

Are other wild or domestic species susceptible to this virus?

Susceptible animals include domestic sheep, horses, dogs, cats, foxes, raccoons, skunks, rats, and mice. Susceptibility of cattle appears to be questionalble and no work had been done to determine the susceptibility of cervids.

Besnoitiosis

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Common Name:

Corn meal disease

Life Cycle:

2 host life cycle:

- definitive host - carnivore
- intermediate host - herbivore

Biting flies have been shown to be involved in the transmission of **B. besnoiti** in South Africa.

	Intermediate	Definitive
B. besnoiti	cattle	cat
B. tarandi	reindeer, caribou	?
B. bennetti	horse, burro	-
B. jellisoni	rodents	cat
+ 4 other **Besnoitia** spp.		

Geographic Distribution:

Besnoitia tarandi

circumpolar	- barren ground caribou, reindeer
British Columbia	- woodland caribou
Saskatchewan	
Manitoba (Assiniboine Park Zoo, Winnipeg)	- caribou, reindeer, mule deer

Clinical Signs:

- often minimal or non-existent
- marked hair loss
- skin thickened, wrinkled, hardened
- secondary bacterial infection/myiasis
- emaciation
- pinpoint ocular cysts

Pathology:

pinhead-sized (1mm) cysts:

- a predilection for lower limbs: connective tissue, skeletal muscle, periosteum
- nasal turbinates

Can be systemic and involve many organs.

Light Microscopy:

Large spherical cysts (90 - 210um in diameter)

Cyst comprised of a peripheral cyst wall surrounding an entire host cell (often multinucleated), probably a fibroblast, within which are large numbers of crescentic bradyzoites (2 - 3um by 10 - 13 um).

Prevalence:

Tuktoyaktuk reindeer:

- 5 - 14% of 200 animals were positive

B.C. 76 of 355 (21.4%) woodland caribou all moose, mule deer negative.

Appears widespread in reindeer and caribou in western and northern Canada.

Apparently transmitted from caribou to mule deer under confined conditions (APZ, Wpg., Manitoba).

Recently found in one muskox in NWT (1988).

Treatment:

None effective.

Sulfonamides, pyrimethamine and arsenicals have been used in cattle in South Africa with limited success.

Potentiated sulfas, amprolium and spiramycin have some effect against other protozoa and could be tried for **Besnoitia** sp. but efficacy not established.

Experimentally, oxytetracycline inhibited cyst development in rabbits.

Domestic Livestock:

Not reported in domestic livestock but there is minimal opportunity.

Infrequent reports of **Besnoitia bennetti** infection in Mexican burros in the U.S.

Research Requirements:

Definition of host range - intermediate and definitive.

Significance of biting flies in transmission of **B. tarandi** must be established.

A research study to attempt to answer this question will be started in July of 1989 in Dawson Creek, B.C.

Group Discussion:

What is the most likely mode of transmission of arctic ***Besnoitia*** *sp.?*

In other species of **Besnoitia**, a cat is usually involved as a final host for this parasite, In the Arctic, there is little opportunity for contact with members of the cat family but other carnivores could be involved. It is tempting to speculate (on the basis of African work as well as preliminary studies at the Assiniboine Park Zoo in Winnipeg) that biting flies may be very important in spreading this infection. Tabanids (horse flies, deer flies) take a large portion of skin when they bite and could effectively transfer infectious material in this manner. Spread of arctic **Besnoitia** sp. by this method, however, remains unproven.

What clinical signs are shown by infected caribou or reindeer?

Clinical signs appear to be non-existent in most cases and, because of this, it is only recently we became aware of **Besnoitia** sp. infection in these animals. We know that animals demonstrating several cysts on the white of the eyes are infected but we do not know how many infected animals do not demonstrate these cysts.

Is there only one type of organism?

Several species of **Besnoitia** have been described based upon the type of animal in which The parasite is found. However, we do not know whether these are, in fact, different species of **Besnoitia**, or the same species in different hosts.

Are blood tests available to detect infection with ***Besnoitia****?*

A serological test is produced in South Africa to detect **Besnoitia besnoiti** but this is not available in North America. We do not know if this test would cross react with **Besnoitia tarandi** in reindeer. In order to detect subclinical infection a serological test must be developed against the specific organism occurring in reindeer. It will probably take about three years to develop such a test.

Should we attempt to eradicate the disease?

It is difficult to eradicate because a serological test is not available to discover subclinical infections and the lack of cysts in the eye does not necessarily mean the animal is not infected. By eliminating from the herd animals with cysts, we can only remove obviously infected animals. If eradication were to be attempted, we must develop a specific, sensitive, reliable and economic test that will detect all infected animals.

How do we determine what other animals may be involved in the life cycle of ***Besnoitia tarandi****?*

It is necessary to culture the parasite in the laboratory to ensure a ready supply of infective material; then infect suspect definitive hosts such as the arctic fox or lynx. An intense research effort must be provided by specialized parasitologists, biologists and veterinarians to determine the life cycle of this parasite.

Parelaphotstrongylus tenuis

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Name of Disease:

Parelaphostrongylosis, cerebrospinal nematodiasis, moose sickness

Elaphostrongylus cervi causes elaphostrongylosis

Type of Disease:

Parasitic

Scientific Name of Parasite and Major Host:

Parelaphostrongylus tenuis (white-tailed deer)

Related forms are:

- **Elaphostrongylus cervi** (red deer)
- **Parelaphostrongylus andersoni** (whitetails, perhaps caribou)
- **Parelaphostrongylus odocoilei** (mule and black-tailed deer)
- **Varestrongylus alpenae** (white-tailed deer)

Common Name of Parasite:

Meningeal worm

Related forms are called meningeal worm (**E. cervi**), muscleworms (**P. andersoni** and **P. odocoilei**) and lungworm (**V. alpenae**)

Known Geographical Distribution:

Eastern and Central Canada including Nova Scotia, New Brunswick, southern Quebec, Ontario, and Manitoba. Eastern and Central United States including Maine, New Hampshire, Connecticut, New York, Pennsylvania, Maryland, New Jersey, West Virginia, Virginia, North Carolina, South Carolina, Georgia, Florida, Alabama, Tennessee, Kentucky, Michigan, Indiana, Wisconsin, Minnesota, Iowa, Missouri, Mississippi, Louisiana, Arkansas, Missouri, Texas, and Oklahoma.

Distribution of related species are as follows: **Elaphostrongylus cervi**; many places in Europe and Asia, New Zealand (introduced), and Newfoundland (caribou). **Parelaphostrongylus andersoni**; southeastern United States and southeastern British Columbia. **Parelaphostrongylus odocoilei**; Alberta, British Columbia, Washington, California.

Clinical Signs:

The white-tailed deer is the normal host for this parasite and it displays few clinical signs. Anderson (1965b) and Prestwood (1970) describe a temporary lameness of the forelimb, and circling and rapid oscillation of the eyeballs, respectively, in white-tailed deer with experimental infections.

In moose and the many other hosts debilitated by infection, the disease is expressed by locomotor incoordination, lameness, stiffness, listlessness, weakness of the hind quarters, circling, abnormal positions of the head and neck, blindness, and paralesis.

Major Gross Pathological Changes:

Extensive lesions of the central nervous system including focal hemorrhaging, neuron degeneration, and yellowish accumiations streaked with blood near worms. Worms are found free in the cranial cavity or on the spinal cord, or partially embedded in nervous tissue. For bare detail see Anderson and Prestwood (1981), Anderson and Strelive (1967), Gilbert (1973), and many others.

Microscopic Changes:

A variety of microscopic lesions have been described (see Anderson and Prestwood, 1981, for details) including small hemorrhages, masses of parasite eggs, infiltrations of white blood cells including eosinophils, and congestion of very small blood vessels.

Method of Diagnosis:

The method of choice is recovery and identification of the adult worm; this of course means that the host must be sacrificed. Recovery of the first-stage larvae in feces using a modified Baermann Technique (see Samuel and Gray, 1982 for a description of this technique) is a step in the right direction, but it must be realized that several other parasitic nematodes of North America shed similar cork-screw and spiny-tailed larvae. These include **Muellerius capillaris** (a parasite of domestic goat and sheep, but also known from bighorn sheep -Pybus and Shave, 1984), **Varelaphostrongylus alpenae** (known from whitetails -Gray et al., 1985), **Parelaphostrongylus andersoni** (in white-tails, see Anderson and Prestwood, 1981; in caribou, Lankester and Hauta. 1989), **Parelaphostrongylus odocoilei** (mule deer, see Platt and Samuel, 1978b), and **Elaphostrongylus cervi** (caribou,see Lankester and Northcott, 1979).

Lengths of some of these larvae differ (Gray et al., 1985) with **V. alpenae** and **M. capillaris** being short and **E. cervi**, long (Gray et al., 198S), but there is much overlap.

Ballantyne and Samuel(1984) differentiated the infective larvae (i.e., the third-stage larvae found in terrestrial snails and slugs) of the three species of **Parelaphostrongylus** using morphology of the tail. Some (Upshall et al. 1986) have found it useful, others (Lankester and Hauta, 1989) not.

Due to the difficulty in diagnosing the infection in live animals, several authors simply recommend that known host species not be translocated from areas where the parasite occurs to new areas.

Method of Treatment and Treatment Effectiveness:

Currently there are no drugs known to be effective against meningeal worm or its close relatives, but little research has been done (Samuel and Gray, 1986).

Other Species Affected by the Same Organism:

Moose, elk, caribou, reindeer, mule deer, black-tailed deer, mule deer- white-tail deer hybrids, fallow deer, red deer, red deer-elk hybrids, domestic sheep, domestic goat, llamas, guinea pigs, and several bovid and cervid species in zoos.

E. cervi has been reported from red deer, roe deer, fallow deer, wapiti, red deer-wapiti hybrids, moose, and woodland caribou.

Prevalence of the Disease in Wild and Domestic Species:

Prevalence of meningeal worm in deer ranges from <1% to more than 85% in various parts of North America. In Canada, prevalence figures for adult worms in the cranial cavities of deer are: 10% (Manitoba), 41-61% (Ontario), 30% (Quebec), 60% (New Brunswick), and 51% (Nova Scotia).

Prevalence of meningeal worm in other hosts is largely unknown although a recent survey in Nova Scotia (Thomas and Dodds, 1988) report it in 6.5% of the moose there. Woolf et al.(1977) report it in from 27 to 64% of a wapiti population in Pennsylvania.

Although prevalence data are hard to come by, there are some interesting semi-quantitative papers (see Saunders,1973). Also, there are many case reports of neurologic disease in moose, some for elk, and up to five reports of disease in other species listed above. Subjective assessments of the importance of the disease abound in the literature. It is probable that serious discussion of this parasite is part of every wild ungulate translocation in eastern North America, because of the documented failures of several reintroduction programs.

Importance to Domestic Species:

Based on the relatively few case reports of disease in sheep and goat from New York, New Hampshire, Connecticut, West Virginia, Ohio, and Texas, this disease is not that important for domestic livestock. However,"the disease may be more common in livestock than is indicated by the literature" (Anderson and Prestwood, 1981). Cattle must have some degree of resistance, at least based on the fact that no cases of disease have been reported.

Public Health Implications:

None known

Research Needed:

Determine whether free-ranging elk of eastern North America shed larvae in their feces. Determine the western/northern/southern range boundaries of meningeal worm and mechanisms delimiting this geographic distribution. Determine susceptibility of various native wild and domestic hosts to meningeal. Develop an effective diagnostic test.

Group Discussion:

Can we be 99% certain that patent infections of meningeal worm can occur in elk?

No, we can be 100% certain that patent infections can occur. Anderson et al. (1966) produced a patent infection in 1 of 2 young calves experimentally infected with meningeal worm. Karns (1966) recovered spined larvae from the feces of wild elk in Minnesota and called them meningeal worm. Pybus et al. (1989) did the same from wild elk in Manitoba, padded them through snails and into 2 white-tails, and recovered adult meningeal worm.

Phase 1 of experiments at the University of Alberta to determine the suitability of elk for meningeal worm is near completion. Researchers infected 10 elk with meningeal worm. The five high-dose animals died of severe neurologic disease; the longest surviving elk in that group shed a few larvae in its feces between day 105 and 111 post-infection. The second-longest surviving elk shed no larvae in its feces, but did have a few larvae in its lungs. All five had numerous mature adult worms. The five low-dose animals showed no signs of clinical disease, and shed no larvae in there feces. At necropsy, three had no worms, one had one adult worm, one had two adult female worms. This project is not complete so that details of the above-presented results are not yet available, but the fact remains that meningeal worm larvae were shed in the feces of an elk.

More studies of wild elk with and without neurologic disease in eastern North America are needed to determine the prevalence of spine-tailed larvae in feces and the identity of any such larvae found. The latter can only be determined by experimental infection of white-tailed deer and recovery of adult meningeal worms.

Will an elk shedding larvae advertise the fact by having severe clinical neurological disease?

This is impossible to answer with certainty at present, although that is what researchers at the University of Alberta found in the one high-dose elk that shed larvae.

Will elk shed larvae in the wild and does it matter?

Two lines of published evidence suggest that they do (Karns, 1966; and Pybus et al., 1989); several line of unpublished evidence suggest that they do not including (Kocan, personal communication; Schmitt, personal communication). The answer is critical because if it could be shown that natural infection does not occur on eastern North America, it would assist with decisions that need to be made in western Canada. Research proposed at the end of the first question above is needed. Also, need are more data on the number of infective larvae available in snails and how deer/elk/moose/etc. become exposed to the snails.

How few meningeal worm larvae in snails dies it take to infect a deer or elk?

There have been no studies on deer or elk related to this question, but it probably takes very few larvae to infect the normal host, the white-tailed deer. Samuel and coworkers at the University of Alberta infected a mule deer with 14 infective-stage larvae of **Parelaphostronglylus odocoilei**, a connective tissue worn associated with muscles that is closely related to **P. tenuis**. The deer remained patent until it was sacrificed at 9.4 years of age.

Several adult meningeal worms were recovered from elk exposed to 15 infective larvae (Samuel, unpublished data)

Are there fluctuations in numbers of larvae shed in the feces?

Yes. Although there are a few if any data for meningeal worm larvae, all studied members of the nematode family Protostrongylidae tend to follow the same patterns. Number of larvae in feces fluctuates by season (more shed in winter-spring than in summer-autumn) and host (the normal or usual host -- in the case of meningeal worm, the white-tailed deer -- tend to shed far more larvae than the susceptible or abnormal host such as elk). Other probable factors influencing fluctuations and levels of shedding of larvae include immunological status and age of the host.

Is the muscle worm similar to ***P.tenuis****?*

The nematode lungworms belong to seven families, one of which (the Protstrongylidae) contains the genus **Parelaphostrongylus** along with 11 other genera. Members of the family Protstrongylidae include many common and important lungworms of ruminants and lagomorphs worldwide. The three members of the genus **Parelaphostrongylus** include: 1) the meningeal worm, **P. tenuis**, associated with the central nervous system of Cervidae (usual host, white-tailed deer); 2) the muscle worm, **P. andersoni**, associated with the intermuscular connective tissue of Cervidae (usual host, white-tailed deer); and 3) the muscleworm, **P. odocoilei**, associated with the intermuscular connective tissue of Cervidae (usual host, mule and black-tailed deer).

What is the known effect of ivermectin on meningeal worm?

In terms of prevention, Kocan (1985) treated deer with 0.1 mg/kg ivermectin at 1, 10, or 30 days after exposure to meningeal worm larvae and only prevented infection in the deer treated 1 day after exposure. In other words, once the larvae had gotten out of the gastrointestinal tract on their way to the CNS, ivermectin had no effect. A second experiment involved four deer treated with ivermectin after the worms had matured and the infection had become patent. Kocan found a reduction in the output of larvae that reached 0 by day 10 following treatment with ivermectin, but larvae reappeared in the feces by day 28 following treatment.

Samuel and Gray (1988) determined that the numbers of first-stage larvae of **P.**

andersoni in the feces of seven white-tails dropped to zero in 17-18 days following treatment with ivermectin ant 200 and 400 ug/kg of body weight. Larvae reappeared in feces 1.5 to 6 weeks later in six deer. Counts dropped to zero in 12 to 18 days in the feces of four of the same deer treated with ivermectin a second time. Larvae reappeared in low numbers 45 to 55 days after the second treatment.

In summary, everyone is expecting that ivermectin is/will be a panacea for worm eradication. In this particular instance, it will probably not be a cure-all because ivermectin does not act across the blood-brain barrier. Multiple dosages of ivermectin over time might be effective, but existing data for similar worms are not promising.

Is meningeal worm a major mortality factor in ungulate populations in eastern North America?

This is a difficult question to answer; many would say yes, some would say no. In both instances, critical data are lacking. What can probably be said with certainty is that meningeal worm is a major pest of moose in many parts of eastern North America. It may have been the reason woodland caribou disappeared from the southern parts of their ranges in eastern North America at the turn of the century. Meningeal worm has been involved in the mortality of many animals such as caribou and elk re-introduced into former ranges in eastern North America. Meningeal worm kills individuals of many species of captive and free-ranging, domestic and wild bovidae and cervidae in eastern North America. The only ruminant species that is common in eastern North America that does not appear susceptible to meningeal worm are cattle.

If it cannot be shown that meningeal worm is an important mortality factor for specific species such as elk, why are we being so sticky on translocating these animals into non-meningeal worm areas?

Meningeal worm is known to be a pathogen for several common native species in western Canada such as moose, elk, mule deer and woodland caribou. Even if it does not devastate populations of these or other species of hosts, it will probably cause thousands of dollars in damage (=deaths, morbidity, paper pushing, responses to public inquiry, lost natural resources, potential threats to domestic animal industry, etc, etc,) annually.

Are current protocols for testing elk for meningeal worm adequate?

Probably not. Consider that infected patent elk probably will not shed many larvae, and they might only shed for a short time. It is not known how accurate the Baermann Technique is when trying to detect low-level infections. The Technique appears to be very sensitive for lungworm larvae such as those of the meningeal worm, but there have been no quantitative studies to determine this.

Perhaps the preliminary results from the experimental study now near completion at the University of Alberta could be used as an example. Only one elk of the five high-dose animals (the low dose produced insufficient numbers of worms to become patent, so they are not considered here) shed larvae and only two others lived past day 90 of the infection (i.e., only three of the five elk survived ling enough to become patent). A total of 7 of 45 Baermann-run fecal samples had larvae, indicating that the ability to detect larvae is either 7/38 (18%) based on the one elk that shed larvae, or 7/45 (based on the three elk that survived more than 90 days).

In contrast to elk, once the low-dose white-tail control began shedding larvae on day 86, only 1/31 (3%) fecals was negative, that on day 87. This deer shed 1 larvae in 30 grams of feces on day 86, 0 larvae in 13 grams of feces on day 87, and up to 136 larvae per gram of feces to day 144 when it was killed. It shed few larvae until day 104 and many larvae between day 104 and day 144.

How would the public respond to existing protocols for dealing with potential importation of meningeal worm into Saskatchewan?

It probably depends on how the scant available information is presented to them. (The question is really not relevant anyway[see next question and answer].) One could envision that the wildlife-interested component of the public would be concerned about introduction of the worm to the native wildlife. Other segments of the public might agree with game growers that the risk is so low as to be of no consequence to animals in Saskatchewan.

Should Saskatchewan monitor elk for meningeal worm as in the past of should new stringent rules be put in place?

This is a risk benefit analysis involving a disease of wildlife that has little involvement with agricultural industry. Let us be clear as to what this means. It means that even though there might be a very small probability of having meningeal worm become established in Saskatchewan, the potential problems/losses that might result could be very high. Also, if meningeal worm is going to cause problems, it is the native wildlife that will be most at risk. If meningeal worm was introduced and became established in Saskatchewan, as happened with rinderpest in Africa when rinderpest-infected cattle were introduced, the wildlife will be impacted negatively at an unknown level. If, on the other hand, steps are taken to avoid the risk of introduction of meningeal worm into Saskatchewan, the game-growing industry will be impacted negatively at an unknown level.

Based on the data collected by the University of Alberta researchers (see shedding rates for elk in above-answer), new more stringent rules for fecal testing may not be any more efficient than the Baermann Technique as employed previously. One possibility for consideration is that elk only be moved onto the province during the probable peak time when larvae are shed (i.e., November to April) thus improving chances of detection.

Frequency of fecal sampling for testing and determining a suitable length of quarantine for incoming elk are also difficult questions to resolve. Based on initial results form the University of Alberta for the three high-dose elk that survived more than 90 days, animals would almost have to be tested daily between 90 and, say, 120 days in quarantine. Not a practical answer, but it is probably the best that can be offered at the moment.

Regarding length of the quarantine, the University of Alberta researchers found that numbers of larvae in feces of an infected white-tail deer did not reach significant levels until approximately 120 days post-exposure.

The only effective answer to this question will come after it has been ascertained whether larval-shedding is common by wild or experimental elk and at what levels.

Given this background information, what are the options?

1) Do nothing while waiting for more information and hope that we do not have a problem.

2) Proceed with caution; follow current protocol.

3) Proceed with caution; quarantine longer, test more frequently.

4) Proceed with caution; quarantine longer, test very frequently. With the latter two options, at least the odds of detecting **P. tenuis** increase.

5) Place a moratorium on importation of elk pending more information or a better monitoring/testing procedure. Some would say that this is difficult to do because of the demand for animals. Others (there is a current moratorium on elk importation into Alberta due to concern about meningeal worm introduction) would suggest that no risk to native free-ranging wildlife should occur no matter what the demand.

Choice of options should probably be influenced by the sober reality that should meningeal worm be introduced into Saskatchewan and become established, it will be extremely difficult if not impossible to eradicate.

Are all hosts equally sensitive to the effects of meningeal worm?

Probably not. White-tailed deer appear to deal with the infection very well and only 2 or 3 deer have been reported with neurologic disease. The literature contains many case reports of natural cases of disease in the abnormal or susceptible hosts. It appears that reindeer, caribou, llamas, and domestic goat are very susceptible. Moose, domestic sheep and elk show some signs of resistance. This area of concern needs to be reviewed thoroughly; there may be enough literature available to categorize host responses.

Are we sure that meningeal worm is not currently in Saskatchewan and if it is not, why not?

Dr. Gray Wobeser, the University of Saskatchewan, and colleagues examined the cranial cavities of approximately 700 white-tailed deer from southeastern Saskatchewan for **P. tenuis** in the late 1970's, early 1980's. All were negative.

In order for meningeal worm (or any parasite for that matter) to survive, several criteria must be met including: 1) presence of adequate numbers and overlapping populations of definitive (white-tails) and intermediate (terrestrial slugs and snails) in sufficient densities to allow for establishment; and 2) a suitable climate for survival of free-living stages of the parasite and suitable numbers of the hosts involved.

If it is not in Saskatchewan, is there a guarantee that it will not be introduced with translocation ranched game animals such as elk?

At present there are no guarantees and few options. If researchers at the University of Alberta find that elk shed meningeal worm larvae commonly in their feces, then a conservative approach would be best for Saskatchewan to close its borders to the importation of elk until more is known about the risk involved. Once host suitability to the worm is determined it remains then to estimate/determine the geographic and climatologic suitability of eastern Saskatchewan for meningeal worm.

What hope is there for development of a more sensitive diagnostic test such as a serologic (ELISA) test?

Although there are no results yet from an initial small pilot project now underway at the University of Alberta, it is highly unlikely that a serologic test can be developed cheaply or quickly. Dr. Mike Belovesic and colleagues are willing to put in the hours, but the research would be expensive. Three years is a time frame given me often by these colleagues, assuming decent levels of funding. Initial results will be forthcoming by mid-September, 1989; after that a more educated guess can be made on the feasibility for development of an ELISA test.

Are there any studies to determine the present geographical distribution of meningeal worm in Canada?

A University of Alberta graduate student is just beginning a study in Manitoba to determine the current distribution and prevalence of infection in white-tailed deer. That study should also address the possible ecological factors that might be involved in influencing meningeal worm distribution.

Flukes

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Name of Disease:

fascioloidiasis, liver rot

Type of Disease:

Parasitic

Scientific Name of Parasite and Major Host:

Fascioloides magna (white-tailed deer, moose, and elk)

Other trematodes reported from wild ruminants of North America include:

Paramphistomum spp. (white-tailed deer, moose)

Zygocotyle lunata (white-tailed deer, moose, elk)

Common Name of Parasite:

liver fluke or the large American liver fluke

Paramphistomum spp. are called rumen flukes; **Zygocotyle** is called the caecal fluke

Known Geographic Distribution:

The large American liver fluke has a spotty distribution in Europe (where it was introduced in the 1750's with American elk) and North America. In the East it occurs in Labrador (Newfoundland), Quebec, around the Great Lakes (including southeastern Manitoba), and in Gulf Coastal States. In the West it occurs in the Pacific Northwest including coastal and Rocky Mountain Trench regions of British Columbia, along the east slopes of the Rockies from westcentral Alberta, Alberta south into Montana, central Saskatchewan. Smaller foci include Cypress Hills (Alberta-Saskatchewan border), Elk Island National Park, Alberta, and in captive population of elk near Lac La Biche, eastcentral Alberta.

Clinical Signs:

Generally none.

Gross Pathological Changes:

Adult flukes occur in fibrous capsules in liver. The capsule walls are whitishyellow, thick and fibrous, often filled with a brownish-black fluid. The surface of the liver is often pitted and contains white fibrous scars. Often, portions of liver surface are adhered to surrounding tissues such as the diaphragm. Immature flukes migrate through liver tissue producing extensive, blood-filled, darkly-pigmented tunnels, surrounded by damaged liver tissue.

Microscopic Changes:

Nothing worthy of detail here but see W. J. Foreyt (1981. Trematodes and Cestodes. In: Diseases and parasites of white-tailed deer [Davidson, W. R., et al., eds.), Miscellaneous Publication No. 7, Tall Timbers Research Station, Tallahassee, Florida. pp 237-265).

Method of Diagnosis:

In the living host that are passing eggs, eggs can be detected in feces using flotation (see Wobeser, G., A. A. Gajadhar, and J. M. Hunt. 1985. **Fascioloides magna**: Occurrence in Saskatchewan and distribution in Canada) or sedimentation (see Sloss, M. W., and R. L. Kemp. 1978. Veterinary clinical parasitology. 5th ed., Iowa State University Press, Ames, Iowa) techniques. One must be cautious because eggs of related trematodes (see above) are similar in morphology to those of **F. magna**. In the dead host, presense of large reddish-brown flukes encapsulated in liver tissue is presumptive evidence of **F. magna** infection, at least in North American hosts such as deer, elk and moose.

Method of Treatment and Treatment Effectiveness:

This is an infection that can be translocated/transplanted/ transported with its host so that preventive measures and/or treatment are important areas of concern. Elimination of snails in vegetated small ponds or prevention of access to such ponds by ruminants will break the cycle. Anthelmintic therapy of liver flukes has been studied extensively and, until recently, there was little cause for optimism because efficacy against immature flukes was limited. The relatively new benzimidazole compound, triclabendazole, used as a 5% drench solution given at 10 mg/kg body weight, has been shown to be effective against adult and immature life stages of liver flukes. A field test of its efficacy in naturally infected elk is underway in Alberta.

Species Affected by the Same Organism:

In addition to white-tailed deer, elk and moose, other vertebrate hosts of **F. magna** include: mule deer, black-tailed deer, red deer, sambar deer, sika deer, roe deer, fallow deer, woodland caribou, bison, yak, chamois, peccary, bluebull, and domestic cattle, sheep and goat.

Prevalence of the Disease in Wild and Domestic Species:

Prevalence of infection in many surveys varies considerably (from <1% to 73%) in white-tailed deer, the most studied host. Prevalence in other hosts includes: 58% (woodland caribou, Labrador), 88% (moose, Minnesota), 86% (elk, Alberta-British Columbia), and 14% (mule deer, Alberta-British Columbia), 37% (cattle, Texas). A total of 1.1 million of 31 million cattle livers federally inspected in the United States in 1973 were condemned due to presence of either **F. magna** or its close relative **Fisciola hepatica**.

Importance to Domestic Species:

F. magna causes fatal disease in sheep and goat and results in liver condemnation in cattle.

Public Health Implications:

Livers damaged by flukes look a mess and should be discarded. Venison from deer, elk, moose, etc., infected with liver flukes is completely safe for consumption.

Research Needed:

Determine better methods of diagnosis in live animals. Determine species of snail intermediate hosts in Baner National Park area. Conduct experimental comparative studies in deer, elk and moose. Determine exact present geographical distribution in western Canada.

Group Discussion:

What kinds of flukes would we expect to find In captive ruminant on game ranches in western Canada?

There are three types: 1) rumen flukes of the genus **Paramphistomum**; 2) cocal flukes (**Zygocotyle lunita**); and 3) the large American liver fluke, **Fasciololdes magna**.

Which of the three appears to be of most concern to game growers?

The liver fluke. Authors of are several papers suggest that it is a sericus peat of moose and elk from certain locales.

Are there carrier hosts or hosts that appear more resistant than others?

White-tailed deer and elk were long considered the ncrmal or usual host for **F. magna**, because 1) the parasite completed its life cycle in those hosts, and 2) the host appeared to tolerate the parasite reasonably well. However, results of recent research in Alberta and British Columbia by Dr. Margo Pybus of the Alberta Fish and Wi:dlife Division suggest that elk are susceptible to massive-level infections and some Individual succumb to the infection.

Domestic sheep are very susceptible to liver fluke infection and the resulting disease is serious in them. Cattle and, apparently, moose show some signs cf resistance. Livers of cattle are condemned. Moose in some areas apparently die as a result of liver fluke-caused disease.

What is the general life cycle of liver fluke and can it be broken easily on a game ranch?

The cycle involves eggs of the parasite being shed in the feces of, say, a deer. If feces are deposited into or near water, the egg can develop into a free-swimming aquatic stage that infects aquatic snails. In snails the parasite develops to a stage that exists the snail and swims to vegetation at the air-water interface, where it encysts on the vegetation. This stage passively infects deer when the vegetation is consumed.

Breaking the cycle involves preventing ungulate hosts from using a water source infested with snails.

Effective drugs include triclabendazole [marketed under the name Fasinex Ciba-Geigy Inc., Grearsboro, NC, USA]. This drug is apparently not cleared for use in Canada at present. A drug called Nilvan is apparently used quite successfully in New Zealand In domestic sheep to control **Fasclols hepatica**, a liver fluke closely related to **F. magna**,

*Can **F. wagna** be detected without sacrificing the animal?*

Eggs can be recovered from the feces of deer and elk, possibly other ranched hosts, using a sedimentation technique. Eggs cf all three flukes mentioned above are similar in morphology. in some hosts such as cattle or moose, eggs are not shed in the feces, because bile ducts passing to the small intestine via the common bile duct are blocked. There is no serological test.

Ectoparasites

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Name of Disease:

Psoroptic mange, psoroptic scabies, scab

Type of Disease:

Parasitic

Scientific Name of Parasite and Major Host:

Psoroptes spp. including **P. cuniculi** (deer) and **P. cervinus** (elk) and **P. ovis** (bighorn sheep)

Common Name of Parasite:

ear or body mite

Known Geographic Distribution:

Rocky Mountains to southeastern United States. Disease in elk is only known from northwestern Wyoming (excluding Yellowstone National Park) and one male elk in northern Idaho.

Clinical Signs:

Usually none.

Gross Pathological Changes:

None in lightly infested animals. In severe cases, especially on bighorn sheep, large accumulations of mites in the ears or on the body result in scroll-like sheets of shed epidermis layered in the hair coat. The ear canal of severely infested animals can be plugged with accumulations of waxy exudate or plugs of necrotic debris. Their ears droop. In elk (the disease is primarily confined to adult males during autumn and winter) the skin (usually the shoulder, chest, and neck) may be covered with dry gray-brown scabs. Purulent exudate, the result of secondary bacterial invaders, is a common feature of the disease.

Micrtoscopic Changes:

Marked lesions only in the skin are characterized by a superficial perivascular dermatitis and severe edema of the epidermis and dermis.

Method of Diagnosis:

Recovery and identification of mites, which is accomplished by scraping superficial skin from obviously infested areas. Samples may be preserved in alcohol (70 - 95%) or formalin (5 or 10%).

Identification to species is virtually impossible; send properly collected mites to a specialist!

Method of Treatment and Treatment Effectiveness:

Treatment of free-ranging animals is difficult, thus the best treatment is prevention through maintenance of good range so that animals remain in good nutritional condition. Captive infested animals such as elk respond well to 200 ug/kg ivermectin given subcutaneously.

Other Species Affected by the Organism:

There are only a few species of the genus **Psoroptes** and little is known about the cross-transmission capabilities of them. **P. cuniculi** has been reported from white-tailed and mule deer, domestic rabbits and goats, blackbuck, ibex, and horses. **P. cervinus** has teen reported from Rocky Mountain bighorn sheep (**Ovis canadensis canadensis**) and elk. **P. ovis** has been reported from desert bighorn sheep (**Ovis c. nelsoni** and **O. c. mexicana**), Rocky Mountain bighorn sheep (**O. c. canadensis**). **P. cervinus** and **P. ovis** must be closely related.

Prevalence of the Disease in Wild and Domestic Species:

In 1982-83, on the National Elk Refuge, Wyoming, essentially the only place where scabies of elk is reported, annual prevalence of infestation was about 26% of the adult males. About 40% of 170 overwinter mortalities were attributed to mange. Scabies is thought responsible for the massive die offs of bighorn sheep herds that occurred in the western United States in the late 1800's and early 1900's (coincident with the arrival of domestic sheep). More recently, sheep scabies has caused mortalities of wild sheep in New Mexico, Arizona, Nevada, and, possibly, Wyoming. Many countries have attempted eradication of "sheep scab", many with only short term success. **P. ovis** was believed eradicated from domestic livestock in Canada and the United States in 1927 and 1953, respectively, but it remains a pest of cattle in several states.

Importance to Domestic Species:

Hard to evaluate due to the lack of information on the cross-transmission capabilities of these mites. Psoroptic mange is an important disease of domestic livestock.

Public Health Implications:

None

Research needed:

Taxonomic work on mite morphology, identification techniques, and cross-transmission capabilities. Determine presence of mites on subclinical animals. Determine the pathogenesis cf the disease. Determine potential presence on mature bull elk in Jasper and Banff National Parks.

Name of Disease:

Acariasis, "ghost moose", tick infestation.

Type of Disease:

Parasitic

Scientific Name of Parasite and Major Host:

Dermacentor albipictus (moose, elk, mule deer, white-tailed deer, horses, cattle). The other tick common on large ruminants of western North America is **Dermacentor andersoni** (found on many ruminant hosts including pronghorn, deer, elk, mountain sheep and goat).

Common Name of Parasite:

D. albipictus; winter tick, moose tick, elk tick

D. andersoni; Rocky Mountain wood tick

Known Geographic Distribution:

D. albipictus; primarily northern and western United States and southern Canada. In western Canada, occurs to 60, North latitude.

D. andersoni; Pacific northwestern United States and adjacent western states. In Canada: British Columbia, Alberta, Saskatchewan and Manitoba.

Clinical Signs:

D. albipictus; loss of winter hair first appears on neck, shoulders, and perianal region], emaciation, loss of fear of man, weakness, and death, (particularly for moose).

Major Gross Pathological Changes:

D. albipictus; skin with extensive rubbed areas, broken and/or lost winter hair, scabbing and local inflammation at point of bite, anemia, malnutrition, loss of body heat.

Microscopic Changes:

No information

Method of Diagnosis:

Visual inspection of skin followed by recovery and identification of ticks (often best left to trained tick taxonomists). The time when adult blood-engorged female ticks are found on or dropping from large ruminant hosts is a clue to identification, because most winter ticks drop from their host in late March-early April before the appearance of the Rocky Mountain wood tick. Mule deer, elk, and moose infected with winter ticks show different yet characteristic patterns of hair loss during winter. This includes: hair loss on all parts of the neck only (mule deer), a notch of hair loss at the base of the dorsal neck region progressing to a collar of loss around the base of the neck (elk), and hair loss beginning on the shoulders, neck, and perianal region progressing to virtually all parts of the animals body except the face and lower legs (moose).

Method of Treatment:

Treatment of free-ranging animals is difficult, thus the best treatment is prevention through maintenance of good range so that animals remain in good nutritional condition. With captive animals a certain way to prevent infestation is to break the cycle; that is, house animals in late March, April, and early May (i.e., when female ticks are dropping from moose or elk) in separate areas from where they will be housed in autumn (when the larvae climb vegetation seeking a host). Spring and autumn fire destroys the engorged female adults and larvae life stages, respecitively. Some game growers have successfully used pesticidal dusts or sprays in early winter (once just after the first lasting snows in November and once 2 - 3 weeks later). ivermectin might work well at this time (i.e., when larvae are feeding), or in February (when the nymph-stage is feeding) or in late March-early April (when the adult-stage is feeding).

Other Species Affected by the Organism:

Not known with certainty for **D. albipictus**. Captive reindeer in western Canada do a lot of grooming against winter tick; some animals have been loaded with ticks. Hair loss on horses with winter ticks has been reported in the literature. There are unconfirmed reports of mild to severe infestations of winter ticks on mountain goats and sheep.

Prevalence of the Disease in Wild and Domestic Species:

Probably infests a high percentage of moose throughout their range, and most elk and deer in western North America.

Importance to Domestic Species:

Except for some information on horses in California and some reported tick-related mortalities of cattle in Saskatchewan in the 1920's, there is not information. Domestic hosts are usually at minimal risk.

Public Health Implications:

None for **D. albipictus**. **D. andersoni** is known to transmit Rocky Mountain spotted fever, Colorado tick fever, tularemia, Q fever, bovine anaplasmosis, tick paralysis, canine piroplasmosis, Japanese type B encephalitis, Lyme disease, etc.

Research Needed:

Determine whether **D. albipictus** vectors other disease-producing agents such as rickettsias. Determine extent of infestation of **D. albipictus** on elk, and also on moose in eastern North America.

Network game-growers in western North America to find out what treatments have worked against ticks on elk or moose.

Name of Disease:

Myiasis, nasopharyngeal myiasis

Type of Disease:

Parasitic

Scientific Name of Parasites and Major Hosts:

Cephenemyia spp. (deer, elk, moose, reindeer). **Cedemagena tarandi** (caribou)

Common Names of Parasites:

Cephenemyia spp. (botflies, deer bots, throat bots). **O. tarandi** (warble fly)

Known Geographical Distribution:

Widely distributed

Clinical Signs:

Harassment of cervids can be extreme during the period when adult flies are depositing eggs or larvae. Animals can behave erratically (caribou sometimes panic), moving about constantly, sneezing and shaking their heads and/or ears as they attempt to avoid being parasitized.

Major Gross Pathological Changes:

Inflammatory reaction within the retropharyngeal recesses that house the large, conspicuous second- and third-stage larvae of **Cephenemyia** spp. Warbles seriously damage the skin of caribou/reindeer causing edema and inflammation to underlying tissues.

Microscopic Changes:

Thickened inflammed mucosa, exudate, and epithelial erosion

Method of Diagnosis:

Recovery and identification of larvae. In most cases, specific identification depends on rearing larvae to the adult fly stage.

Method of Treatment and Treatment Effectiveness:

None known for botflies of deer, elk or moose. Successful control of warbles and botflies in reindeer herds in Alaska involves use of an injectable organic phosphate product (Warbex) repeated yearly during the period between September and January when the warble larvae are small and located in the host.

Other Species Affected by the Same Organism:

None

Prevalence of the Disease in Wild and Domestic Species:

No data known for warble of **Rangifer** in North America. Prevalences of botfly larvae range up to 95% in deer.

Importance to Domestic Species:

Apparently, none

Public Health Implications:

Apparently, none

Research Needed:

Evaluation of pathogenecity of botfly larvae for hosts infested experimentally.

Name of Disease:

Louse infestation

Type of Disease:

Parasitic

Scientific Names of Parasites and Major Hosts:

Tricholipeurus parallelus (white-tailed and mule deer), **T. lipeuroides** (white-tailed and mule deer), **Bovicola** (=**Damalinia**) sp. (elk, mountain goat and mountain sheep)

Common Name of parasite:

Chewing or biting lice

Known Geographical Distribution:

Probably occurs throughout the geographic distribution of hosts.

Clinical Signs:

None

Major Gross Pathological Changes:

Usually none. Hairloss can occur in animals that are grooming in response to the lice.

Microscopic Changes:

None reported.

Method of Diagnosis:

Visual insfection of skin (inguinal and axillary regions) followed by recovery and identification of lice. Specimens should be preserved in 70% alcohol and sent to a louse taxonomist for confirmation of identification.

Method of Treatment and Treatment Effectiveness:

Chemical treatments used successfully against lice of domestic livestock should work on captive wild ruminants.

Other Species Affected by the Same Organism:

Chewing lice are generally quite host specific; consequently, it is rare that other hosts are affected by the same organism. Captive blackbuck of Texas have been found infested with chewing lice (**T. parallelus**) of whitetailed deer.

Prevalence of the Disease in Wild and Domestic Species:

Very rare. Mentioned here because under conditions of crowding and poor nutrition (such as could occur in captive situations), lice can become a problem.

Importance to Domestic Species:

None

Public Health Implications:

None

Research Needed:

Renewed taxonomic work on identification of lice from elk, caribou, and bison.

Appendix I.

Some important of potentially important* ectoparasites of wild ruminants with appropriate references.

	Common Name	Scientific Name	Appropriate Reference
ELK	Scab Mite	**Psoroptes pervinus**	1,2,3
	Winter Tick	**Dermacentor albigictus**	4
	Mange Mite	**Sarcoptes scabiei** (red deer only)	18
BISON	Louse	**Damalinia sedecimdecembrii**	19, 20
REINDEER	Warble Fly	**Oedemagena tarandi**	5
	Nasal Bots	**Cephenemyia trompe**	6
	Winter Tick	**Dermacentor albipictus** (acquired)	
WHITE-TAILED and MULE DEER	Ked or Louse flies	**Lipopena** and **Neolipoptena** spp.	7,8,9
	Chewing Lice	**Tricholipegrus** spp.	10,11,20
	Bot Flies	**Cephenemyia** spp.	12,20
	Lone Star Tick	**Ambdyomma americanum**	4,13
MOOSE	Winter Tick	**Deramacentor albipictus**	14,15,20
MUSKOX	have no known ectoparasites		
MOUNTAIN SHEEP	Scab Mite	**Psoroptes ovis**	1,3,16
FALLOW DEER	Lice	**Bovipola libialis**	17

*Capable of seriously impairing the health of the host.

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Anaplasmosis

Submitted by L. Schofield

Synonym:

Gall Sickness

Game Species:

White-tailed deer, Mule deer, Pronghorn Antelope, Columbia Black-tailed deer, Elk, Bighorn Sheep, Bison.

Type of Disease:

Anaplasmosis is a rickettsiale disease. In the broader sense of the term could be considered bacterial. In domestic cattle the etiologic agent **Anaplasma marginale** causes an acute infection characterized by fever, depression, emaciation, severe debilitation, progressive anemia due to erythrophagocytosis and icterus with no accompanying hemoglobinuria.

In wild ungulates of North America is considered subclinical.

Scientific Name:

Anaplasma marginale

Geographic Distribution:

Considered a tropical/subtropical disease. Commonly found in cattle of South Africa, Australia, U.S.A. and U.S.S.R. with the potential of southern Alberta, Saskatchewan and central portions of British Columbia.

Inter-mountain region, Montana, Wyoming, Colorado, Utah, Southern Saskatchewan, Alberta, and Central B.C. is the range of **Dermacenter andersoni**, commonly called the "Wood Tick" potentially significant.

Inter-mountain region offers semi arid short grass condition needed for tick transmission.

- Transmission occurs with appearance of the vector.
- Modes of transmission ticks, flies, iatrogenic transplacental
- Significance coulees and watering holes?

Clinical Signs:

In domestic cattle signs are:

- Fever
- Depression
- Weight Loss
- Constipation
- Anemia
- Jaundice
- Abortion
- Reduced Exercise Tolerance

In wildlife signs are:

- Most wild ungulates are susceptible to experimental infection, but do not show signs unless Splenectomized.
- Columbian black tailed deer are the only cervids reported to have shown clinical disease.
- Symptoms similar to domestic cattle
- Mortality insignificant amongst infected ;wild ruminants.
- Susceptibility varies with age and condition.

Gross Pathology:

- severe anemia
- tissue pallor
- jaundice
- splenomegaly - reddish brown pulp, enlarged follicles hepatomegaly - yellow colour depending on degree of icterus
- distended gall bladder - dark grummous bile
- pericardial petechiation

Histopathology:

- presence punctate inclusion bodies margin RBC
- bone marrow hyperplastic with extra medullory haematopoiesis
- presence of reticulocytes in prepheral circulation.

Diagnosis:

	*CF	CTA	RCA	MRCA	IFA
W T D	+	+		+	N/A
	problem false positive	fewer false positive			
M D	-			+	N/A
B T D	-		+	+	N/A
ELK	-		+	+	N/A
PRONGHORN	-			+	N/A
BIGHORN	-			+	N/A
BISON	+		+	+	No Data

*CF -- can be misleading with numbers of false positive and negatives.

DNA probe methods appear to be the most accurate method of diagnosis as well as for strain identification.

Treatment:

- treatment infrequent due to subclinical infection. If required oxytetra cycline parenterlly 7 - 11 mg/kg for 3 - 5 days.

- due to stress aspect a reposital form should be given the risk of treatment, by handling, may exceed the disease risk.

- carrier state eliminated by feeding medicated feed 7mg/kg daily for 60 days.

Control:

- no effective vaccine to date

- test and slaughter

- tick/fly control?

Other Species Affected:

	A. marginale	**A. ovis**	**A. centrale**
Bovine	+++	+ 177,262d.	++ mild
Ovine	+ no clinical signs	++ mild	-
Caprine	+	++	-

Cross reactions occur in cattle & Sheep using A. marginale and A.ovis antigen

	A. marginale	**A. ovis**	**A. centrale**
Species			
W.T.D.	+ nat exp	+ exp	
M.D.	+ nat exp		
B.T.D	+ nat exp	+ exp	
ELK	+ exp	+ exp	
PRONGHORN	+ exp		
BISON	+ exp		
BIGHORN	+ exp		
F.D.	+		
R.D.	+		

Other African and Asian wild ungulate species can be experimentally infected causing subclinical infectious with A. marginale, A. ovis and centrale

Significance of these infections unknown

threat to domestics unknown

Prevalence of Anaplasmosis in Wild and Domestic Species:

- Anaplasmosis does not exist to our knowledge in any wild or domestic animal in Canada

- considered exotic and reportable in Canada

Importance to Domestic Species:

- national herd susceptible

- cattle 3 years and older - acute disease

- direct genetic, reproduction and calf losses could have

high impact on cattle industry.

- high cost test and slaughter policy

- may effect export markets

- no effective vaccine

- endemicity would cost millions annually.

Zoonotic Implication:

- none

Research Needed:

Canadian strain identification if in fact such a thing exists.

Seroprevolence surveys in wild cervid populations in southern Saskatchewan and Alberta

Tick population dynamics study.

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Group Discussion:

Is one age group of bovine more predisposed to infection?

Generally calves under a year of age manifest subclinical or mild disease which causes little set back. Animals 1-2 years of age manifest acute disease that is rarely fatal; animals between 2 and 3 years experience acute disease that is occasionally fatal while cattle over three years most often suffer peracute disease that is often fatal.

What is the expected geographic range of naturally occurring anaplasmosis in Canada?

Since 1967 anaplasmosis has been introduced into Canada three times. The first two intrusions occurred in Manitoba in 1967 and Quebec in 1979. The third was in Saskatchewan in 1983. The first two introductions were accidental importations. The 1983 occurrence was a naturally occurring outbreak in southern Saskatchewan. The natural occurrence of a disease is always associated with the presence of the agent, that is the causal organism, a host able to sustain it and an environment that favours its' growth. The agent, **Anaplasma marginale** is positionally contiguous to all the western provinces by virtue of it's natural occurrence in the American states in apposition. The typical biome (environment), supporting the agent, comprising semi-arid short grass prairie, exists in most of southern Saskatchewan, Alberta and British Columbia. Finally a suitable host exists in the large cattle populations found throughout this area along with the suspected vector, the wood tick, **Dermacenter andersoni**. These factors place at risk southern areas of Saskatchewan, Alberta and potentially the intermontane areas of British Columbia supporting wood tick populations. Since the necessary bioclimatic components do not exist in the central or northern portions of these provinces, it is most likely outbreaks will be confined to the areas described.

Does wildlife play a part in spreading or harbouring this disease?

In Canada there is no indication at this time, that wild ungulates such as deer, antelope or other smaller prairie mammals such as wild canids, ground squirrels, porcupine, rats or mice play any role in spreading or sequestering anaplasmosis.

What other ways could anaplasmosis potentially spread?

There are two ways of concern. The first is veterinary practices such as castration, vaccination, dehorning, tagging or any minor surgical procedure that produces a blood flow. If instruments are not sterilized between animals or disposable instruments used, the risk of infecting any number of animals is there. It is recommended instruments be either sterilized between animals or a separate disposable instrument such as syringes, scalpels, taggers and dehorning wires be used.

The second concern is certain biting flies and mosquitoes. In the southeastern United States as well as other parts of the world where anaplasmosis is present, documented infection occurs by mechanical transfer on the infected mouth parts of these insects. Canada has the fly and mosquito species which are able to effect these transmissions. It is not known at this time if these insects play an active role in the spread of this disease. It is certainly not outside the realm of possibility. It must be mentioned that these flies and mosquitoes occur in the pacific and central northwest USA as well but have not been shown to have an impact on the transmission of anaplasmosis. To date, the wood tick, **D. andersoni**, is implicated as the major vector and it is assumed, although not fully substantiated, this is the vector in southwestern Alberta and Saskatchewan. The reason this assumption is made is the terrain and climate for both these areas is virtually identical. More studies are needed to bear this out.

What is the reservoir of greatest concern in Canada?

Undoubtedly, the domestic bovine is the major reservoir of disease, for both a clinical source of re-infection to a susceptible host or as a quiet carrier that may go undetected yet able to infect herd mates under the right conditions.

What is the best protection against anaplasmosis?

There is no one practice that will protect cattle against anaplasmosis. To date there is no effective vaccine. The use of fly tags and prophylactic doses of wermectin are helpful in reducing fly and tick loads. Careful consideration should be given to sterile techniques when dehorning, castrating, vaccinating, spaying and ear tagging. Animals should be purchased from clean sources. This should be true for all herds in Canada as we are considered free of anaplasmosis.

What should I do if I suspect symptoms of anaplasmosis?

You are obligated, under law, to contact your nearest federal district veterinarian. He or she can be found in the phone book under Government of Canada, Agriculture Canada, Food Production and Inspection Branch, the Veterinary Inspection Directorate. An examination and diagnosis will be made immediately to determine if anaplasmosis is present.

Malignant Catarrhal Fever

A report compiled for the Saskatchewan Technical Game Farming Advisory Committee
Submitted by J.E.G. Smits D.V.M., WCVM.
University of Saskatchewan

Malignant catarrhal fever (MCF) is a disease of ruminant animals that has been seen in Africa[23], Asia[5,22], Europe[16,19], Canada and United States[6,7,16,18,] Australia[16] and New Zealand[1,12]. It is a clinicopathological syndrome primarily affecting the gastrointestinal and upper respiratory tracts, eyes and lymphoid organs. This disease is characterised histologically by necrotizing vasculitis of the small and medium sized arteries in most organs. MCF usually has low morbidity, (although there have been epizootics involving bovine and cervine species)[7,14,22], but clinical cases are almost invariably fatal.

Etiology:

There are two recognized forms of the disease, the African form, or wildebeest associated(WA)-MCF, and sheep associated (SA)-MCF in most other affected areas of the world. The WA-MCF has an identified aetiological agent. What was formerly called bovid herpesvirus 3 is now called alcelaphine herpesvirus 1 It is inevitably associated with the presence of alcelaphine antelope including white-tailed gnu, hartebeest and blue wildebeest, so outside Africa this form of MCF is seen in zoological parks or exotic animal game preserves. Elsewhere in the world where the SA-MCF form is seen, the aetiological agent remains a mystery.

Epidemiology:

WD-MCF is thought to be an infectious cell free virus, which is less sensitive to humidity and temperature than cell associated virus. This viral form would be important for transmission in natural conditions[4]. The virus has been isolated from the ocular and nasal secretions of healthy wildebeest calves up to three months of age[13]. The alcelaphine herpesvirus 1 has been successfully isolated from white tailed gnu and bearded gnu without clinical signs of the disease[9]. Virus has also been found in the buffy coat of blood from neonatal and 12 day old wildebeest calves[4]. From all these findings it is clear that wildebeest (**Chonnochaetes spp.**) act as reservoirs and latent carriers of WD-MCF.

The SA-MCF is thought to be spread by sheep into the environment, carried by human vectors, by effluent and by direct contact between sheep and susceptible species[14]. A history of previous contact with sheep is almost invariably established when a case occurs. The agent is thought to be a normal resident of sheep, which is excreted during the periparturient period[19].

New Zealand studies have shown the disease to be stress associated. Among farmed deer on the south island, 50 % of winter deer deaths were due to MCF. There was a higher incidence on intensively (vs. extensively) managed properties1, and twice as many males as females were affected. These parameters concur with the stress theory; males enter the winter quite worn thin from the rut, higher animal density adds to social stress and the winter season's cold and wet weather is an additional problem for the deer.

Clinical Signs:

The clinical signs associated with MCF vary with species and the type of MCF. With WA-MCF the majority of cases are characterized by a marked febrile response after a variable incubation period. Depression, photophobia, lacrimation, salivation, corneal opacity, upper GIT and muzzle erosions and crusting are seen in bovidae and cervidae. Lymph node enlargement is usually palpable[6,19,23].

With the SA-MCF, cattle may develop the head and eye form, the peracute form or the alimentary form of the disease[12]. The head and eye form is the most commonly seen in cattle, while diarrhoea and dysentery are seen in cervids[17]. This disease in cattle differs from the African form in that the incubation period is thought to be considerably longer, 32 days, while the disease course is an average of 3.5 days, and it is more frequently accompanied by diarrhoea[16]. In red deer, affected animals have been found dead, or have developed keratitis, diarrhoea and dysentery and died after two or three days[12]. Rarely, more chronic cases have been observed in which the animals live from four to 13 days.

There are numerous species which have been seen with MCF; white-tailed deer are usually found dead[24,17], moose, pronghorn antelope and reindeer have been found with typical MCF lesions[17], and Pere David deer die peracutely or within three days with muzzle crusting, ocular lesions and dysentery[14]. Interestingly, wapiti in the same conditions as the Pere David deer did not show signs of MCF. They do get it, but less commonly than red deer, sika deer and Pere David deer, from observations at the Invermay Animal Health Research Center in N.Z.[14]. There have not been any cases of MCF described in fallow deer at this time!

Gross Pathology:

Pathologic lesions of MCF are very similar between the two disease 'types', and do not provide a basis for distinction[19]. The WA-MCF presents upper gastrointestinal tract erosions, crusts and ulceration, from the muzzle down to and including the abomasum[6]. There is a generalised lymphadenopathy most remarkable about the head and neck, and nasal turbinate necrosis with tracheal congestion and ecchymosis. Many cases have had renal cortical patches of pallor due to leukocyte infiltation and infarction[6,23].

The SA-MCF in deer in the United Kingdom has produced necrotizing lesions of the upper respiratory and gastrointestinal tract epithelium, along with hypertrophied lymphnodes[14], while in other areas the most consistently found lesions have been hemorrhagic, nectrotizing enteritis and typhlitis (cecum inflammation)[19,24]. Dramatic large bowel pathology is often the only visible lesion in peracute cases[19], while more chronic cases have urinary bladder petechial hemorrhage and multiple pale raised renal foci[11,19]. Bovine cases may have the lesions described above and commonly are seen with bladder and upper GIT pathology[10].

Histopathology:

Histopathological vascular lesions are pathognomonic for MCF[2,4,19]. The brain appears to be the most consistently affected organ, with renal lesions being almost invariably present also[14,19]. In the brain, the tunica media and perivascular space of the medium sized arterioles are infiltrated by mononuclear cells. This CNS vasculitis is especially prominent in the leptomeninges and cerebral white matter[6,14]. Perivascular infiltration is accompanied by smooth muscle, connective tissue and endothelial cell degeneration. Vascular fibrinoid degeneration and necrosis is pathognomonic in ruminants[14,17].

In more chronic cases, there is marked meningeal endothelial cell proliferation. In numerous tissues including lungs, liver and kidney, there is a mild interstitial mononuclear cell infiltration[14]. In the liver, lymphocytic accumulation is most consistently associated with vessel walls in the portal triads and subcapsular regions[11,23]. In the upper respiratory and gastrointestinal tract, there is epithelial cell degeneration, along with lymphocyte, plasma cell and macrophage invasion of the lamina propria[6].

Diagnosis

The most useful and widely used tools for the diagnosing of MCF are clinical signs with histopathological confirmation (because the cases are almost invariably fatal)[6,23]. Confirmation by virus isolation or neutralization is only possible yet for WA-MCF. Virus identification can be done either by electron microscopy[4], or by the typical cytopathic effects on bovine embryonic kidney cell cultures of the buffy coat cells of clinically affected individuals[4,16,23]. Immunofluorescent techniques are used to demonstrate either intranuclear inclusion bodies or the cytopathic effects which produce massive syncytia[4].

The detection of antibodies to WA-MCFV (alcelaphine HV-I, formerly bovid HV-3) is achieved either by indirect immunofluorescence (IIF) tests or indirect immunoperoxidase (IIP) technique. A comparison of these tests indicated that the IIP was more sensitive in antibody detection than IIF when used on clinical cases[21]. In order to rule out false positive cross reactions with other bovine herpesviruses, testing sera at 1:100 dilution in the IIF test improves its accuracy[9]. Attempts to detect the SA-MCF agent have produced some reaction with alcelaphine HV-I by IIF but nothing by virus neutralization (VN).

Treatment

Treatment of this condition is considered hopeless. There have been rare cases of recovery from clinical MCF, but it is accepted to be greater than 90 % case fatality[14]. The only reasonable action would be the isolation of the affected individual, and attempting to identify and remove the original source of the infection.

Species Affected

Ruminants have been the main group to be involved in natural cases of MCF. This clinicopathological syndrome causes different mortality and morbidity patterns depending upon where in the world it is occurring and which species are involved. on the whole there is low morbidity and very high mortality. SA-MCF has caused sporadic losses in white-tailed deer, axis and mule deer, moose and reindeer[17] in the United States, sika deer in Ontario[23], white-tailed deer in Saskatchewan[24] and wapiti and bison in Alberta, according to a disease survey conducted by the WCVM pathology department. Cattle are affected occasionally anywhere MCF occurs. Domestic rabbits and hares have been experimentally infected, and have shown the same clinical and pathological changes as are seen in ruminants, but natural infection has not been seen.

In zoological collections in North America and world wide, many species of captive wild ruminants have been affected by WAMCF. Situations when there have been more than rare sporadic cases, generally involve special environments such as zoos or large exotic game ranches. Prevalence in domestic and wild species in Canada is very low. The Ontario VSL has approximately 3 cases of MCF per year, B.C. diagnoses it less than once per year, Manitoba and Saskatchewan have had rare cases in zoo ungulates, according to the afore mentioned survey.

In New Zealand where the host species of SA-MCF is so numerous, MCF has been the major source of lossed in farmed deer in four of the past five years[1]. In Australia, MCF among farmed deer is seen in sheep grazing regions, while in the cattle grazing areas it is rarely seen.

In "outbreak" situations, natural deer to deer transmission has been suspected in N.Z. and in Canada[12,24] and has been shown to be possible experimentally in the U.K.[20]. However, there was no deer to deer WA-MCF transmission among experimentally infected deer which were run together with noninfected deer[23].

Conclusion:

The multiple challenges remaining to SA-MCF researchers yet, include demonstration of the aetiologic agent, knowledge of factors contributing to its pathogenicity and the identification of all the potential carrier species. Lambing sheep have been well established as a major epidemiological prerequisite to SA-MCF[10,14,19,22].

With all the information from MCF epidemiology elsewhere in the world, we in Canada are in a good position to maintain the incidence at the presently very low level. Continuous flow of information, particularly to people who will be involved in deer farming is a sound policy to avoid problems similar to those that have arisen in N.Z. although low numbers of domestic sheep do dramatically reduce the risk here.

Group Discussion:

Are free ranging mountain sheep vectors of MCF?

There have begn no reported cases of MCF in wild sheep species in Canada or the U.S. They are not thought to be a reservoir of SA-MCF. Other big game species which have had MCF (WTD, sika deer, moose, wapiti, bison and reindeer) have been in captive or semi-captive management systems.

Does an animal dying of WA-MCF act as a source of infection to others?

Non host animals with WA-MCF die within days of developing clinical signs. They are called "dead end" hosts, and have not been suspected of transmitting the disease.

What is the major threat associated with SA-MCF?

The infective agent has not been identified yet, despite many efforts by researchers to isolate it.

The infection has been transmitted from sheep during the lambing season, to vulnerable populations of deer. "Outbreaks" of MCF have been described in these herds, and deer to deer transmission has been suspected, but never proven.

SA-MCF is of much greater concern to us than WA-MCF for the reasons stated above, but also became sheep are much more likely to come into contact with game farmed animals, than are the alcelaphine antelopes (wild beast).

Why is the disease such a major problem to the deer farming industry in N.Z. despite research efforts on SA-MCF?

MCF is one of the top two disease problems there, because of that country's enormous sheep industry. Direct or indirect contact (though effluent or vectors) is difficult to prevent. Lack of industry support has put restrictions on research.

Are there specific species resistant to SA-MCF?

Fallow deer may be resistant to SA-MCF because there are no reports of them having the disease. Wapiti in N.Z. show a greater resistance than some other deer species. Bighorned sheep are not known to get MCF perhaps because of inherent resistance of sheep species.

What would you recommend to game farmers with respect to MCF contact?

Never raise deer with, adjacent to, or within effluent runoff distance from, domestic sheep. Do not put deer onto paddocks which have had sheep on them, within the past few months. Be strict about quarantine of deer imported from New Zealand.

Bovine Tuberculosis

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Type of Disease:

bacterial

Scientific terms:

Mycobacterium bovis

Other species of mycobacteria, especially **M. avium**, can produce lesions similar to those of bovine Tuberculosis. These other mycobacterial infections are noteworthy because of their potential to confuse the interpretation of tests and the detection of bovine tuberculosis.

Common Names

tuberculosis (TB)

mycobacteriosis

Geographical Distribution:

Worldwide. Many countries have TB control programs but few have entirely eliminated the disease. Eradication of bovine tuberculosis from the livestock population of Canada is very near completion. The last known reservoir of bovine tuberculosis in Canada is the free-ranging, hybrid bison population in and around Wood Buffalo National Park.

Clinical Signs:

The clinical signs of tuberculosis vary with the distribution of the lesions and the severity of the disease. Tuberculosis is often manifested as a chronic wasting disease of individual animals within a herd. Respiratory tract lesions can give rise to dyspnea, coughing, oral and nasal discharge, and exercise intolerance. Superficial lymph nodes may become noticeably enlarged and may develop draining sinuses. occasionally, as in miliary tuberculosis, the disease may progress so rapidly that few clinical signs are seen before the animal is found dead. In herds depopulated because of tuberculosis, some individuals that appear to be in good health will often have extensive, severe lesions at necropsy. Hence, the clinical diagnosis of tuberculosis can be very difficult.

Major Gross Pathological Changes:

Lesions of tuberculosis vary from small, focal granulomas in individual lymph nodes, to extensively disseminated, coalescing granulomas involving many organs systems. These granulomas are usually pale lesions, often well circumscribed and expansive, with caseous centers that may vary in consistency from semi-fluid ("suppurative") to hard (mineralization and fibrosis). Mycobacterial infections cause a spectrum of disease depending on the species and health status of the infected animal. Therefore the appearance of the lesions can vary both within and between host species.

Microscopic Changes:

Tuberculosis produces granulomatous lesions. Generally there is a focus of caseous necrosis surrounded by macrophages, epithelioid cells, lymphocytes, fibroblasts and variable numbers of giant cells. Acid-fast bacilli can usually be found within the necrotic debris, macrophages and giant cells of these lesions.

Neutrophils may be present in the lesions in some instances.

Method of Diagnosis:

The gross and histological characteristics of the lesions, with presence of acid-fast bacilli, provide a tentative diagnosis. Definitive diagnosis depends on bacteriological isolation of the agent from lesions or exudate. Intradermal skin testing, using suitable tuberculin(s), may provide strong evidence of infection. However, skin test results can be unreliable or difficult to interpret depending on the host species, physiological status of the animal, and the stage of the disease. Therefore, more reliable information can be obtained from testing a herd rather than an individual animal.

Methods of Treatment and Effectiveness:

Despite extensive research, no reliable method of treating tuberculosis in animals has been found. No effective vaccine have yet been developed. Control of the disease is based on test and slaughter.

Species Affected by the Same organism:

Tuberculosis can occur in humans and a very broad range of domestic, wild and exotic animals depending on circumstances of exposure. However, under natural conditions, tubercle bacilli are generally maintained in a limited number of primary hosts, which are the reservoirs of the disease: **M. tuberculosis** in primates; **M. bovis** in bovids; **M. avium** in birds. In game farmed species, bovine tuberculosis has been reported in elk, bison, reindeer, white-tailed deer, mule deer, moose, fallow deer, sika deer, red deer, marals, axis deer, roe deer, mouflon, wild boar, llamas and yak. The disease has also caused significant mortality in foxes and mink on fur farms. Close confinement and the stress of captivity are major factors in the occurrence and maintenance of tuberculosis in host species not normally associated with the disease.

Clinical Signs in Other Species:

As mentioned above, the spectrum of disease can be highly variable within and between host species.

Prevalence in Wild and Domestic Species:

Tuberculosis is quite rare in freeranging wildlife in North America -- with the exception of the bison population in Wood Buffalo National Park, Canada. However, tuberculosis can be a significant cause of morbidity and mortality in captive wildlife populations, eg. zoo stock, game farmed deer. Tuberculosis is becoming increasingly uncommon in livestock in several countries because of control and eradication programs. However, few countries have succeeded in eradicating bovine tuberculosis.

Importance in Domestic Species:

Tuberculosis is a serious disease of domestic animals, and thus most nations have implemented control and eradication campaigns. Tuberculosis is a reportable disease in the Animal Disease and Protection Act of Canada. Outbreaks of tuberculosis can have serious national and international repercussions for the marketing of livestock and livestock products. This is equally true for game farmed animals.

Public Health Implications:

Bovine tuberculosis is a zoonotic disease. Farmers, veterinarians, and slaughter house workers have contracted the disease, or become positive on Mantoux test, from handling infected livestock and farmed deer and elk. The disease can also be spread to people via consumption of raw milk from infected cattle.

Research Needs:

Research efforts are currently being directed at evaluation of conventional TB tests in a broader range of host species, and development of new tests with the hope of improving the accuracy and convenience of casefinding programs.

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Group Discussion:

Which tests for tuberculosis work well?

Intradermal skin testing is not perfect, but is the best system that we have at this time. The literature suggests that the caudal fold test (PPD tuberculin) has a sensitivity approaching 90%, and a specificity of approximately 95%, when used in cattle. However, these values can be lower when the caudal fold test is used in other species. When cervical testing has been used in cattle surveys, usually the single (bovine PPD) cervical test has been used. Experimental and field trials suggest that the cervical test, single or comparative (bovine and avian PPD), is the test of choice for use in farmed deer. Sensitivity and specificity data indicate that we can have about the same degree of confidence (no more, no less) in cervical tests in red deer and caudal fold tests in cattle. It has been noted that very careful preparation of the cervical injection site is critical when using this test in deer species.

Extensive efforts have been made to try to develop a good serological test for TB in animals and, especially, in humans. Enzyme-linked immunosorbent assays (ELISA) have received much recent attention and, while some look promising, they have not yet reached the point where they can stand alone as officially sanctioned tests. They are being evaluated as adjunct or back-up methods to intradermal test. Lymphocyte transformation tests have, and are, being evaluated but need further work before they might prove to be reliable.

Is there a problem with certain tests used on animals entering Canada?

The answer depends upon the species of animal in question. The caudal fold test appears to be as reliable in bison as it is in cattle. The caudal fold test does not seem to be as reliable when used in cervids. Different tests may have to be done on different species; research may be necessary to determine if disparities exist between tests in different species. It is important that the people doing the test be well trained in the procedure so that the results are uniform.

What percentage of animals in a group should be tested?

It is too risky to only test a percentage of animals in a group, especially in light of the potential consequences of missing an infected animal. Sampling theory is dependent on the suspected prevalence of the disease and the number of animals in the population of interest. If the prevalence is very low, the percentage that need to be tested becomes very large if you hope to detect the disease. Regarding the risk, tuberculosis has often gone undetected in a herd until extensive lesions were found in the sentinel case and a high percentage of the animals were infected. The disease can spread extensively before clinical cases are detected.

When **M. bovis** is detected in a herd, that herd is placed under quarantine; test reactors in a confirmed infected herd are killed and disposed of (eg. burial), and the remainder are sent to the abattoir. Deer that are TB tested in the U.S. for export to Canada are subsequently retested in Canada. The need for uniform record keeping was emphasized so that trace-backs can be done effectively, and sales of animals followed, if TB were to occur on a game farm.

Bovine Brucellosis

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Type of Disease:

bacterial

Scientific Terms:

Brucella abortus

The species has been divided into 8 biovars (groups with distinctive biochemical characteristics). These biovars are useful epidemiological markers for following the spread of the disease between populations of animals.

Common Names:

bovine brucellosis, Bang's disease, contagious abortion, undulant fever (humans)

Geographic Distribution:

Worldwide. Most countries have control programs, but few have succeeded in eliminating the disease. Canada's cattle population was declared free of bovine brucellosis in 1985. The hybrid bison population in and around Wood Buffalo National Park is the last known reservoir of bovine brucellosis in Canada. Although many states in the U.S. are free of brucellosis, the disease is still a problem in cattle and commercial bison operations in a few parts of that country. Also, the bison and elk populations of Yellowstone National Park, and elk populations in Wyoming, have a high incidence of the disease.

Clinical Signs:

Brucellosis impairs reproductive performance by causing abortion, metritis, orchitis, seminal vesiculitis, and epididymitis. Offspring may be born alive but in poor condition. Brucellosis can also cause arthritis, spondylitis, bursitis, and tenosynovitis resulting in lameness and reduced mobility. Asymptomatic carrier animals are common, especially in chronically infected herds.

Major Gross Pathological Changes:

In abortion cases, there is usually a pronounced placentitis with necrosis of the cotyledons, leathery plaques in the intercotyledonary spaces, and edema. Uterine lesions (metritis) are not pathognomonic for brucellosis. Lesions of orchitis range from multifocal dranulomas to abscessation. Granulomas also occur in the epididymides and seminal vesicles. There can be extensive atrophy and fibrosis of the testicle(s) and accessory glands in chronic cases. Non-suppurative synovitis with hygroma development can occur, especially in carpal and stifle joints. These lesions can progress to a severe, erosive arthritis.

Microscopic Changes:

While noteworthy, the microscopic changes in the placenta, fetus, male and female reproductive tracts, periarticular tissues, joints, and lymph nodes are generally not pathognomonic for brucellosis.

Method of Diagnosis:

Definitive diagnosis of brucellosis depends on the bacteriological isolation of brucellae from lymph nodes, reproductive tract (fetus, placenta, uterus, uterine fluids, testicles, epididymides), and joint lesions (synovium and synovial fluid). Serological testing is the major means of detecting the disease in animal Populations. A wide range of serological tests have been developed. In Canada, the tests of choice include the standard tube agglutination test (STAT), buffered plate agglutination test (BPAT), complement fixation test (CFT), and card test. The brucellosis ring test (BRT), conducted on milk samples, is also used in the surveillance of cattle populations in Canada and elsewhere. No one test will detect brucellosis in every instance, therefore a battery of tests is usually recommended.

Method of Treatment and Effectiveness:

There is no reliable means of eliminating the bacteria from infected animals. A vaccine (Strain 19) is available for control of bovine brucellosis in cattle, but it is only about 70% effective and will not, by itself, eliminate the disease from an infected herd. Experimental studies have indicated that Strain 19 vaccine, even at low dosages, is much more pathogenic in bison than in cattle. Test and slaughter has been the method of choice for eliminating brucellosis from Canada. Bovine brucellosis is a reportable disease in the Animal Disease and Protection Act of Canada.

Species Affected by the Same Organism:

Brucella abortus can sporadically produce antibody titres or infection in humans and a wide range of wild and domestic animals, depending on the circumstances of exposure. However, **B. abortus** has a relatively restricted range of primary reservoir hosts, mainly bovid species: cattle, bison, water buffalo, African Cape buffalo, yak, etc. Antibody titres have been reported in camelids (camels, llamas, vicuna, alpacas) but it is not clear what role, if any, these animals play in the epidemiology of the disease. Sporadic cases of brucellosis in other species are often due to close association with an infected, primary host species. The occurrence of **B. abortus** in elk populations in Wyoming is the result of intensive winter feeding programs which attract and condense large numbers of elk at the time of year when abortion is most likely to occur. The disease has not been detected in free-ranging elk elsewhere in the U.S. or in Canada. Other intensive game management systems, such as game farming, may also produce unnatural circumstances which favour the transmission and maintenance of brucellosis in species which normally would not be true reservoirs of the disease.

Clinical Signs in Other Species:

As listed above. The very few published reports of brucellosis in moose suggest that it is a severe, often fatal, disease in this species.

Prevalence in Wildlife and Domestic Species:

In Canada-and the U.S., there are only two significant wildlife reservoirs of bovine brucellosis: the bison in and around Wood Buffalo National Park, and the bison and elk in the Yellowstone/Wyoming region. The incidence of the disease is high in these populations, based on serological surveys. The disease has been eradicated from Canadian livestock. It does occur in a small percentage of cattle herds in some states in the U.S.

Importance to Domestic Species:

Bovine brucellosis is a very significant disease of cattle. Great efforts are taken to prevent the reintroduction of this disease into Canadian cattle. The disease is reportable, and is subject to eradication whenever it might occur.

Public Health Implications:

Bovine brucellosis is an important zoonotic disease, referred to as "undulant fever" in people. Symptoms include bouts of fever, headache, muscle pains, and general malaise. Diagnosis of the disease can be difficult and, if untreated, the disease can periodically recur months or even years after the initial infection. A small percentage of human cases develop more severe lesions which include valvular endocarditis, meningitis, orchitis, and arthritis.

Research Needs:

Research is currently aimed at development of more sensitive, inexpensive, and rapid diagnostic tests. Programs must be established to limit the risk of transmission of brucellosis from problem wildlife reservoirs (free-ranging bison and elk) to livestock and captive wildlife populations.

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Group Discussion:

Now that Canada's livestock population is free of bovine brucellosis, the level of surveillance testing is winding down. Should we still be testing game farmed species coming into Saskatchewan from other parts of Canada?

Although the cattle population in Canada is free of brucellosis, the status of all populations of game farmed animals has not yet been determined. Unmanipulated, freeranging elk populations in Canada are not reservoirs of brucellosis, but elk in close confinement on game farms are a potential reservoir. There has been a high incidence of brucellosis in bison herds in the U.S., and the disease was found in a bison herd in Ontario in 1988. Consequently, there are good reasons to continue testing game farmed species moved between provinces.

Dr. E.T. Thorne noted that a battery of tests, rather than any single test, should be used for greater consistency in detecting brucellosis in elk. No one test will detect every case of the disease. Evaluation of data indicates that the group of serological tests used to detect brucellosis in Canadian cattle are equally effective when used on elk.

What will Agriculture Canada do if it finds seropositive animals on a game farm?

The situation would be handled the same way as it would in a cattle operation. The reactor animal(s) would be retested to ensure that the serological evidence was solid and, if so, the reactor(s) would be slaughtered for bacteriological evaluation. If Brucella abortus was isolated, the herd would be quarantined and depopulated.

What are the chances that infected animals may slip through the tests?

Odds are not high, but there is always a slim possibility of .a very recently exposed animal or a latent carrier being test negative. The chance of letting these individual cases "slip through" can be significantly reduced by knowing the history of the herd of origin and results of brucellosis testing in that population. Latent carriers were defined as calves, born to infected dams, that do not test positive until some time after they reach sexual maturity. The routine tests used to detect bovine brucellosis (**B. abortus**) in Canada will also detect seroconversion due to exposure to the other smooth brucellae, i.e. **B. suis**, **B. melitensis**, and **B. neotomae**. These tests will not detect infections caused By rough brucellae, **B. ovis** or **B. canis**.

Brucellosis Caused By **Brucella Suis**

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Type of Disease:

bacterial

Scientific Terms:

Brucella suis

The species has been divided into 5 biovars (groups with distinctive biochemical characteristics and, in some cases, distinct host preferences).

Common Names:

brucellosis of swine, porcine brucellosis; rangiferine brucellosis of caribou/reindeer

Geographical Distribution:

Worldwide. Biovars 1 and 3 occur in domestic and feral swine in most countries that raise pigs. Biovar 2 occurs in domestic swine, wild boar, and hares in Europe. Biovar 4 occurs in caribou and reindeer in Canada, Alaska and the U.S.S.R., but not in Scandinavia. Biovar 5 occurs in rodents in northern areas of the Soviet Union. In Canada, the domestic swine population is free of brucellosis; the only known sources of **B. suis** are barren ground caribou and reindeer (**B. suis** biovar 4). Biovars 1 and 3 exist in domestic and feral swine populations in the U.S.

Clinidal Signs:

Infected swine are often asymptomatic carriers. The disease can cause fetal reabsorption and abortion in gilts and sows; this can occur in individual animals or, less frequently, as outbreaks involving several sows. Piglets may be born alive but in poor condition. Infected females frequently develop granulomatous metritis,which can cause reduced reproductive success or sterility. Boars develop lesions in testicles and accessory glands which may result in reduced fertility. **Brucella suis** can also cause arthritis resulting in lameness, or spondylitis that can result in paralysis. In caribou and reindeer, **B. suis** biovar 4 causes poor reproductive success due to abortion, metritis, orchitis and epididymitis. It also causes debilitation due to arthritis, tenosynovitis and bursitis in these hosts.

Major Gross Pathological Changes:

In swine, the uterine lesions of **B. suis** range from a catarrhal endometritis to Imiliary uterine brucellosis" (characterized by numerous yellowish nodules or abscesses, which may coalesce into plagues, in the mucosa). In abortion cases, the placenta may be congested and edematous and the fetus may have blood-tinged edema fluid in the subcutaneous tissues and body cavities. Boars develop granulomas and caseous "abscesses" in testicles, epididymi, and accessory sex glands. These lesions may result in atrophy and fibrosis of these structures. Joint lesions in the limbs and spinal column range from synovitis to purulent or fibrinopurulent arthritis which may lead to caseous necrosis of bone and destruction of intervertebral disks. **Brucella suis** is an more aggressive pathogen than other species of brucellae and can occasionally produce focal granulomatous lesions in a variety of visceral organs and lymph nodes. Lesions of **B. suis** biovar 4 in caribou and reindeer are similar to those caused by p. suis biovars 1,2, and 3 in swine.

Microscopic Changes:

Microscopic changes corresponding to the gross lesions are notable, but are generally not pathognomonic for **B. suis** infection.

Method of Diagnosis:

Definitive diagnosis depends on bacteriological isolation of the causative agent from lymphoid, reproductive, articular or other tissues of an infected animal. A wide range of serological tests have been developed. In Canada, the same tests used to detect bovine brucellosis in cattle are used to detect **B. suis** infections in swine, caribou, and reindeer (standard tube agglutination test, buffered plate agglutination test, and complement fixation test).

Method of Treatment and Effectiveness:

There is no reliable method of eliminating the bacteria from infected animals, and there is no effective vaccine for use in swine. Scientists in the U.S.S.R. and Alaska have been trying to develop an effective vaccine for use in reindeer and caribou. Most countries attempt to control or eradicate the disease in domestic swine through test and slaughter programs.

Species Affected by the Same organism:

Brucella suis infections can occasionally occur in other species that are in close contact with true reservoir hosts (domestic and feral swine, wild boar ! caribou, reindeer). These other species likely do not play a significant role in the transmission of the disease. The occurrence of the disease in feral swine or "wild boar" has complicated efforts to control the disease in the U.S.. Serological and/or bacteriological evidence of brucellosis in humans, sled dogs, wild carnivores (wolves, foxes, bears), and muskoxen in northern Canada and Alaska have been attributed to contact with infected caribou and reindeer. The potential infectivity of **B. suis** biovar 4 for other Wildlife species in temperate climates has not been evaluated.

Clinical Signs in Other Species:

There is little information on the nature of the lesions, if any, produced by suis infection in wildlife other than the primary host species. Rodents often die when experimentally infected. **Brucella suis** biovar 4 has caused arthritis and orchitis in muskoxen.

Prevalence in Wildlife and Domestic Species:

Brucella suis biovar 4 Appears to be enzootic in most barren ground caribou herds in Canada. The brucellosis status of woodland caribou and Peary's caribou populations is not known. There has been serological evidence of infection in the reindeer herd near Tuktoyaktuk, N.W.T. Other varieties of **B. suis** do not occur in Canada.

Importance to Domestic Species:

Most countries, especially those with significant investment in the swine industry, consider this to be an important disease, and have established brucellosis control programs to prevent the spread or import of **B. suis**. The disease can cause orchitis in dogs and bursitis in horses. In Canada, it is a reportable disease in swine. Results of very limited experimental studies suggest that **B. suis** biovar 4 does not produce disease in cattle, sheep or swine, but will cause seroconversion in tests for brucellosis.

Public Health Implications:

Brucella suis infections in people range from asymptomatic to severe clinical disease. In rare instances, it can cause death. The organism is more virulent than **B. abortus**, and can produce widely disseminated, caseous granulomas in a variety of organs. A small number of cases of brucellosis, due to **B. suis** biovar 4 infection, in Inuit people are reported annually in northern Canada.

Research Needs:

Research is needed to determine if **B. suis** biovar 4 affects livestock, game-farmed wildlife, or free-ranging wildlife in southern Canada. This should be done before infected reindeer are translocated from the arctic to other parts of Canada. Surveys should be done to determine the brucellosis status of commercially harvested caribou populations in Canada.

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Group Discussion:

Drs. E.T. Thorne and A. Kocan noted that Canada should pay particular attention to brucellosis testing when importing swine/wild boar. Brucellosis in feral swine is a significant problem in the U.S. One person suggested that perhaps Saskatchewan should not allow imports of wild boar. Others suggested discouraging game farmers from raising wild boar. It was noted that swine imported into Canada are tested for brucellosis.

Brucellosis Caused By **Brucella Ovis**

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Type of Disease:

bacterial

Scientific Terms:

Brucella ovis

Common Names:

ovine brucellosis, ram epididymitis

Geographical Distribution:

Worldwide, in countries producing sheep.

Clinical Signs:

Rams develop scrotal edema and inflammation followed by enlargement and fibrosis of one or both epididymides and testicular atrophy. These changes result in reduced fertility or sterility. Only in New Zealand has the disease been reported to cause abortion and birth of weak or stillborn lambs.

Major Gross Pathological Changes:

Early in the course of infection there is swelling and edema of the scrotum with inflammatory exudate around the testicles, and granulomas in the epididymides and testicles. In more chronic cases, the epididymis is firm and enlarged and the testicles have undergone atrophy. In cases of abortion, the placenta is edematous with thick, leathery yellow plaques in the intercotyledonary spaces, and there is necrosis and enlargement of the cotyledons.

Microscopic Changes:

The microscopic features of the granulomatous lesions in the epididymides and testicle, and necrotizing placental lesions are not pathognomonic for **B. ovis** infection.

Method of Diagnosis:

Several serological tests have been developed for surveillance of brucellosis in sheep. In Canada, the complement fixation test is the method of choice. Serological tests used to detect **B. abortus** and **B. suis** infections in livestock will not detect **B. ovis** infections in sheep, and vice versa. Rams suspected of having the disease may have palpable lesions in the epididymides and testicles. A definitive diagnosis is based on isolation of **B. ovis** from semen, aborted fetuses and placentas, or other tissues.

Method of Treatment and Effectiveness:

Antibiotic therapy and vaccination methods have not been thoroughly evaluated and are not used to control the disease in Canadian sheep. In some countries, test and slaughter is used to control or eradicate the disease.

Species Affected by the Same Organism:

Naturally occurring cases of **B. ovis** infection appear to be restricted to domestic sheep. Experimental infections have been induced in goats and white-tailed deer. A noninfected white-tailed buck did become infected and did develop epididymal lesions after being kept in close confinement with an infected buck. This suggest that under unnatural conditions of close confinement, deer might be able to maintain and transmit the disease.

Clinical Signs in Other Species:

Experimentally infected goats and white-tailed deer developed granulomatous lesions in epididymi, similar to those described in rams.

Prevalence in Wild and Domestic Species:

The disease is not reported to occur in wildlife or domestic species other than domestic sheep. The disease is present in some flocks of domestic sheep in Canada, but the incidence appears to be low. The possible significance of this disease in species of wild sheep has not been evaluated.

Importance to Domestic Species:

The disease is of importance to domestic sheep, and appears to be restricted to this species. The economic impact of the disease can be significant, and is a special concern of major sheep producing countries. Although "brucellosis" is a reportable disease in Canada, there is no program in place to control or eradicate **B. ovis** in the Canadian sheep population.

Public Health Concern:

nil

Research Needs:

It would be useful to know if the disease can be maintained and transmitted in species of sheep other than the domestic varieties.

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Yersiniosis

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Type of Disease:

bacterial

Scientific Terms:

Yersinia pseudotuberculosis(formerly **Pasteurella pseudotuberculosis**)
Yersinia enterocolitica bacterial

Common Names:

yersiniosis, pseudotuberculosis (a variety of other names have been used in the past, but precise etiology was often uncertain or attributable to other agents)

Geographical Distribution:

Worldwide

Clinical Signs:

Animals are often just found dead. When signs have been documented, they include diarrhea, dehydration, toxemia/septicemia, depression, loss of body condition, and rough hair coat. In addition to causing enteric disease, **Y. pseudotuberculosis** infection in domestic animals (cattle, sheep, goats) has caused abortion, mastitis, epididymitis and orchitis. Subclinical infections also occur.

Major Gross Pathological Changes:

Lesions vary to some degree between host species, but usually involve the intestinal tract and associated lymph nodes. The small intestine, cecum, and large intestine may all be involved to varying degrees, and lesions range from severe congestion (with or without hemorrhage), to necrosis and ulceration of the mucosa (with fibrinonecrotic pseudomembrane in some instances). Mesenteric lymph nodes are usually enlarged and congested or edematous. The peritoneal fluid may be blood-tinged and may contain fibrin. Miliary abscesses or pyogranulomas can develop in a wide range of lymph nodes and organs due to hematogenous and lymphatic showering of bacteria from the intestinal tract. Yersinia infections have also been associated with both septic and non-septic arthritides in some host species, and with abortion, mastitis, epididymitis and orchitis in livestock.

Microscopic Changes:

Histologically, the appearance of the miliary abscesses varies with the age of the lesions. The nodules originate as foci of caseous necrosis, which become surrounded by macrophages and neutrophils. Colonies of **Yersinia** sp. can be seen around the margins of the necrotic debris. Mineralization can be seen in older lesions and the surrounding cell population is more typical of a granulomatous reaction (macrophages, epithelioid cells, fibroblasts, lymphocytes). Giant cells are notably absent from these lesions.

Methods of Diagnosis:

Bacteriological isolation of the organism from lesions or from feces of clinically affected animals is required for a definitive diagnosis. Both **Y. pseudotuberculosis** and **Y. enterocolitica** can be recovered from the feces of asymptomatic hosts, and **Y. enterocolitica** has been isolated as an "opportunist or secondary invader" from lesions *attributed to other agents* (eg. malignant catarrhal fever in farmed deer). Serological tests have been developed, but are not widely available for veterinary purposes. Antibody titres are reportedly short-lived in affected animals.

Methods of Treatment and Effectiveness:

Broad spectrum antibiotics have proven to be effective in the treatment of yersiniosis if administered early in the clinical course of the disease. Tetracyclines, chloramphenicol and tribrissen have been successfully used to treat deer in New Zealand. Penicillins and streptomycin are said to be less effective in treating deer. Antibiotics have been used in healthy deer as a prophylactic measure to limit or prevent outbreaks of the disease. Because the occurrence of the disease is usually predisposed by stress, efforts to reduce or eliminate stress factors may reduce the prevalence and consequences of the disease.

Species Affected by the Same Organism:

Yersiniosis has been reported in humans and in a very wide range of domestic, wild, and exotic mammals and birds. Regarding game farmed species, **Y.pseudotuberculosis** infections have been reported in elk, white-tailed deer, mule deer, fallow deer, roe deer, red deer, muskoxen, mouflon, and wild boar.

Clinical Signs in Other Species:

As listed above.

Prevalence in Wild and Domestic Species:

There is little accurate information on the prevalence of infection in most species. The disease usually only receives attention during sporadic outbreaks in livestock, or occasionally-recognized epizootics in wildlife. Carrier animals have been detected in many animals populations where the disease was unsuspected. Yersiniosis has been an economically significant disease of farmed deer in New Zealand and Britain. Epizootics have occasionally occurred in fur-farmed species (chinchilla, mink, martin, fox, nutria).

Importance to Domestic Species:

Yersiniosis due to **Y. pseudotuberculosis** has been documented in swine, cattle, sheep, goats, horses, rabbits, cats, dogs, chickens, and turkeys. However, the disease is not common in domestic animals and, therefore, does not cause significant economic loss to the livestock industry. Sporadic outbreaks of the disease, notably in sheep, can raise local concern. Despite the rarity of the disease, the organism can often be isolated from feces of clinically normal livestock.

Public Health Concern:

Yersiniosis is a zoonotic disease. It can be manifested as enteritis, "pseudoappendicitis" (RLQ syndrome), mesenteric lymphadenitis, septicemia (sometimes fatal), arthralgia, polyarthritis, and erythema nodosum. Yersiniosis has occurred in deer farmers in association with outbreaks in captive deer in New Zealand.

Research Needs:

Documentation of management procedures aimed at preventing or reducing the manifestation of yersiniosis through the reduction of stress in captive animal populations.

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Group Discussion:

Do wild birds carry yersiniosis?

Wild birds and rodents have been found to carry yersiniae, and have been blamed as a source of infection. However, it is interesting to note that in outbreaks of yersiniosis, serotypes of the organisms do not always match between adjacent human and animal populations. Game farming literature from New Zealand indicates that tetracyclines are the drugs of choice in treating yersiniosis.

Dr. J.C. Haigh noted the seasonality of the disease. In New Zealand, where the disease is a problem in farmed deer, outbreaks of yersiniosis usually occur in the winter months. Red deer bulls appear to be most susceptible just after rutting season. In muskoxen on Banks Island, the outbreaks occur during the summer. The seasonality of the disease, and increased prevalence in post-rutting bulls, likely reflect the periods of greatest stress on the animals; stress is a major predisposing factor to clinical yersiniosis.

How often can human cases of yersiniosis be traced back to wildlife sources?

There are many different serotypes of **Y. pseudotuberculosis** and **Y. enterocolitica**; serotyping is an invaluable tool in mapping transmission of the disease. In some instances, yersiniosis in humans has been convincingly traced to animal sources of infection. However, in other instances, multiple serotypes have been involved with no clear correlation between those in people and those in domestic and wild animals.

Paratuberculosis

Thorne/Williams

Species:

Ruminants

Agent:

Bacterial (**Mycobacterium paratuberculosis**)

Scientific and Common Names:

Paratuberculosis, Johne's disease

Known Geographic Distribution:

Paratuberculosis is found throughout North America, but is generally uncommon in areas with a relatively dry climate such as the intermountain west and the southwest US.

Clinical Signs:

Clinical signs early in the course of the disease may be very subtle and hard to detect. The most common clinical sign in all species is loss of body condition to a state of emaciation with continued normal appetite. A second common clinical sign is chronic diarrhea, though in some mountain sheep, mountain goats and deer, diarrhea may not appear until the terminal stages of the disease. Affected animals are usually alert and responsive and do not have a fever. The hair coats of affected animals are often rough. Duration of clinical disease is prolonged, with diarrhea and loss of condition occurring over a period of months. Paratuberculosis is a disease of adult animals due to the long incubation period. Morbidity is usually low; mortality is high.

Gross Pathology:

Gross lesions of paratuberculosis are variable and may be subtle or striking. If the animal had diarrhea, feces are often present on the hocks and perineum. The animals are usually in poor body condition and muscle atrophy may be present. Enlargement of mesenteric lymph nodes, especially those draining the ileum, and the ileocecal lymph nodes are commonly observed in animals dying of paratuberculosis. Less frequently, there is enlargement of the wall of the distal small intestine, cecum, and spiral colon. Lymphatic draining the intestine are often thickened and opaque and mesenteric edema is present.

Microscopic Changes:

The presence of the bacteria induces a granulomatous inflammatory response. Granulomas and/or diffuse granulomatous infiltrations occur in the mucosa and submucosa of the distal small intestine, cecum, and spiral colon, lymph nodes associated with the intestine, and lymphatic draining affected intestine. Lesions may be found occasionally in tonsil, lymph nodes of the heads liver and pancreaso and in the liver and spleen. Acid-fast stains frequently demonstrate numerous bacteria within epithelicid macrophages and giant cells.

Methods Of Diagnosis:

Diagnosis of paratuberculosis in the living animal is difficult due to the variable response of individuals infected with this pathogen. A definitive diagnosis requires isolation of the bacteria by culture of the feces; this is seldom very practical as the bacteria grows very slowly requiring as long as 16 weeks. A variety of serologic tests have been developed for use in domestic animals; few have been tested in nondomestic species and currently none are considered very accurate. Serologic tests which are being developed for domestic animals (enzyme linked immunosorbent assay, radioimmunoassay, etc.) may be useful for diagnosing paratuberculosis in nondomestic animals in the future. Lymphocyte blastogenesis tests are fairly accurate in mountain sheep but not in cervids and the test is difficult to conduct and not widely available. The intravenous johnin test has not been tested in nondomestic species and is not likely to be practical due to the need to determine serial body temperatures. Intradermal testing is not reliable.

Diagnosis of paratuberculosis in animals at necropsy is based on gross and microscopic changes which may be characteristic; however, individual animals may vary in the degree of lesion formation. Definitive diagnosis of paratuberculosis requires culture and isolation of the organism. Tissues which should be submitted for culture include distal mesenteric lymph nodes, ileocecal lymph nodes, ileum, ileocecal junction and cecum. Refrigerated or frozen tissue is suitable for culture.

Treatment:

There is no treatment for animals affected with paratuberculosis. Prevention of the disease is the most important aspect of control and is accomplished by limiting new additions to a captive herd to animals obtained from herds free of the paratuberculosis. Contact with domestic ruminants of unknown paratuberculosis status should be limited. The disease is insidious due to the presence of subclinical carriers and the long duration of incubation during which bacteria are shed in the feces. Vaccines have not been widely used in domestic animals and have not been tested in nondomestic animals. The lack of good diagnostic tests for the disease in nondomestic animals makes control of paratuberculosis very difficult. Animals with clinical signs should be eliminated from the herd and feces from all animals should be cultured. Those animals shedding the organism should be eliminated; fecal cultures of all animals at six month intervals should be continued until there are three negative tests. Sanitation is important as the organism is shed in the feces; however, the bacterium is very resistant to environmental conditions and can persist in moist dark conditions for several years. Drastic measures to control paratuberculosis, including complete depopulation, have been used in herds of captive wild sheep

Other Species Affected:

All ruminants are probably susceptible to paratuberculosis. Transient infections, which are probably not important in the epizootiology of the disease, have been reported in swine and horses.

Clinical Signs In Additional Species:

The clinical signs of paratuberculosis in all ruminant species are similar; however, the occurrence of diarrhea may not be a prominent feature of the disease in some species, particularly sheep and goats.

Prevelance In Wild And Domestic Animals:

Prevalence of paratuberculosis is variable but appears to be uncommon in dry climates. Infection is more common than disease and many infected animals never develop clinical paratuberculosis. The disease is common in dairy cattle and some states and breed associations have instituted control programs. Paratuberculosis appears to be less common in the beef breeds of cattle and in sheep and goats than in dairy cattle. Surveys of clinically normal wild deer living in areas where paratuberculosis was common in cattle showed about 10% infection rate. Paratuberculosis has been reported from free-ranging deer, elk, mountain sheep and mountain goats, though the paucity of reports of clinical disease in these species suggests that the disease is uncommon. Paratuberculosis is probably more common in captive nondomestic ruminants and has been reported in a variety of species and from a variety of management situations including zoos and game farms.

Importance To Domestic Species:

Paratuberculosis is an economically important disease to the dairy industry and control programs have been instituted by some states. It is of less importance to the other domestic ruminant industries; however, the disease can have considerable negative impact on individual producers.

Transmission:

The causative bacteria is shed in large numbers in the feces. Other animals may then ingest the organism through fecal contamination of feed. Infected animals, who may be clinically normal, may shed the organism in the feces for months or years and thus are a menace to susceptible animals sharing the same environment. Young animals appear to be more susceptible to infection than adult animals. Congenital infections may occur but this method of transmission is less important than the fecal-oral route.

Public Health Implications:

Humans are not considered susceptible to paratuberculosis. Recently, however a bacteria similar to **M. paratuberculosll** has been isolated from a few cases of chronic enteritis in humans. Good sanitary practices, including hand washing and avoiding fecal contamination of food for human consumption, should be practiced.

Research Needed:

Development of sensitive, specific, and practical diagnostic tests is the primary research need.

Group Discussion:

Does Johne's disease occur in Saskatchewan?

Johne's disease probably occurs in domestic sheep and cattle in Saskatchewan. It is not currently a reportable disease in the Province. (Note - you should check locally on the distribution of Johne's disease in Saskatchewan)

What is the distribution of Johne's diseases across Canada?

Johne's disease is endenic in Canada and may occur frequently in exotic wild animals.

Would it be possible to eradicate Johne's disease?

Johne's disease would be very difficult to eradicate for several reasons. Presently there are no effective diagnostic tests and there is no treatment for the disease. The bacteria that causes Johne's disease is very hardy in the environment and it is difficult to adequately clean premises that have housed infected animals. Another problem has to do with the very long incubation period during which animals may appear healthy and normal even though they may be shedding the arganisn into the environment. Detection of clinical disease in game animals may be even more difficult because they usually do not develop the profuse diarrhea that characterizes the disease in domestic cattle. Close confinenent of animals as in a game ranching situation probably increases the possibility of transmission of the disease between animals.

What problems could game ranchers experience if they had Johne's disease in their animals?

There would be a loss in productivity of the aninals which developed clinical signs and probably a 2 - 5% mortality from the disease. The cost of attempts at control would be expensive and might require loss of valuable breeding stock. Other game ranchers would not want to purchase animals from the infected herd.

Is there a reliable way to avoid bringing Johne's disease into a herd?

No, because some dealers are unscrupulous. Always inquire about the disease exposure status of any animals you purchase and always purchase from reputable dealers or ranches. Do not purchase any animal from a zoo or ranch that has had Johne's disease. The tests for Johne's disease are often not reliable in gane animals so don't rely solely on negative test results.

How can the Johne's disease status of herds be determined?

Johne's disease is not a reportable disease so information on it's occurrence is difficult to obtain. Documentation of disease occurrence would be valuable and the disease should probably be made reportable. When the disease is confirmed in an animal facility this information should be made known to the game ranching "network". However, accurate diagnosis which means necropsy of suspected cases and confirmation by growth of the organism is of great importance. Inaccurate diagnosis and hearsay could be a serious financial hardship on a game farmer.

How difficult is it to make a diagnosis of Johne's disease in game animals.

Diagnosis of Johne's disease may be very difficult in game animals. The clinical disease in some animals and some species may be subtle and consist of a gradual weight loss rather than severe diarrhea. Necropsy findings may also be subtle in comparison to the marked lesions in mesenteric lymph nodes and intestine that are common in affected domestic animals. The disease must be confirmed by isolation of **Mycobacterium paratuberculosis** from tissues or feces of the affected animals, because some other mycobacterial species may cause sporadic cases of disease that may resemble Johne's disease.

Contagious Ecthyma

Thorne/Williams

Species:

Mountain Sheep, Mountain Goat

Scientific And Common Names:

Contagious ecthyma, sore mouth, scabby mouth, scab mouth

Known Geographic Distribution:

British Columbia, Alberta, Alaska, Wyoming, Colorado, New Mexico; likely to occur wherever domestic sheep and mountain sheep or mountain goats occur.

Clinical Signs:

Mountain sheep and mountain goats with contagious ecthyma experience intense itching and appear restless, nervous, and show increased licking of lips and nostrils. The disease is characterized by scabby proliferative lesions on the lips, nostrils, eyelids, and occasionally, teats. Scabby lesions may occur on the gums and hard palate. Presence of scabby lesions makes grazing and suckling difficult; females with painful teat and udder lesions may not allow lambs or kids to nurse. Consequently, weight loss frequently accompanies contagious ecthyma. Recovered animals may be left with irregular depigmented areas where scabs occurred on the nose and lips.

Gross Pathological Changes:

Proliferative, elevated, and scab covered areas occur on the lips, nostrils, udder, and teats. A raised cauliflower-like surface, which bleeds in the live animal, is revealed upon removal of a scab. Lesions may become confluent in severe cases and cover the entire mucocutaneous junction of the mouth and nares. Secondary bacterial infections may occur, and are characterized by suppurative exudate. Contagious ecthyma rarely causes death; but when it does, the animal will be emaciated.

Microscopic Changes:

Histologic examination reveals well delineated epidermal areas characterized by foci of epithelial cells experiencing balloning degeneration. The basal epidermal layer is highly proliferative with peg formation into the dermis containing numerous mononuclear cells, edema, and increased vascularity. Secondary bacterial infections are accompanied by microabscesses in the acanthotic epithelium and neutrophilic response. Transient eosinophilic cytoplasmic inclusion bodies may occur, but they are rarely present after eight days of infection.

Methods Of Diagnosis:

A presumptive diagnosis of contagious ecthyma can be based upon clinical signs and presence of scabby lesions in affected sheep or goats. Complement fixation and serum neturalization tests can be conducted on blood serum from affected or recovered animals. The pox virus can be demonstrated in scab material by electronmicroscopy.

Treatment:

There is no treatment for the pox virus that causes contagious ecthyma. However, supportive treatment consisting of antibiotic administration against secondary bacterial infection and nurtritional supplementation may be helpful. Contagious ecthyma is highly contagious and use of common feeding structures, watereres, or salt blocks should be discouraged if there is concern about transmission to other animals. Vaccines are available for domestic sheep and goats. These vaccines consist of live virulent virus which produces small active lesions that result in protection against reinfection. Because the vaccines consist of live virus, their use must be carefully considered as they may cause active infection in associted animals and environmental contamination.

Because contagious ecthyma is so highly contagious, attempts to control or stop an outbreak in a susceptible captive population may be impossible. It might be more practical to allow the disease to run its course and provide supportive treatment when necessary. The virus is very resistant in the environment and may survive many years in dried scab lesion material. Phenol disinfectants and steam kill the virus.

Other Species Affected:

Mouflon sheep, barbados sheep, ibex, muskox are susceptible. Experimental artificial infections have been produced in mule deer, white-tailed deer, elk, moose, caribou and pronghorn antelope.

Recently, poxvirus infections resulting in dermatitis, keratoconjunctivitis, and lesions on the velvet have been described in mule deer, reindeer, red deer, and pronghorn. These diseases are still being studied, but their occurrence should alert the game manager to the possibility of unusual poxvirus infections in their stock.

Clinical Signs In Additional Species:

Clinical manifestations of contagious ecthyma in mouflon sheep, barbados sheep, ibex, and muskox would be similar to those seen in domestic sheep and goats and mountain sheep and mountain goats. Clinical signs in artificially infected cervids and pronghorn antelope were mild.

Prevalence In Wild And Domestic Species:

Contagious ecthyma is common in domestic sheep, and goats, and it can be expected to occasionally occur wherever mountain sheep and mountain goats are confined or occupy habitat in close proximity to domestic sheep.

Importance to Domestic Species:

Outbreaks of contagious ecthyma in domestic sheep and domestic goats results in economic loss due to weight loss by affected animals, orphaning of occasional lambs and kids, and rare deaths.

Transmission:

Spread of contagious ecthyma occurs by direct contact with active lesions on affected animals or scab material in the environment. Contact with contaminated inanimate objects in the environment by susceptible animals may result in transmission.

Public Health Implications:

Contagious ecthyma is easily transmitted from affected animals to humans, and it is called orf in humans. Contagious ecthyma in man results in localized painful skin lesions that spontaneously regress after three to four weeks.

Research Needed:

The importance and affects of contagious ecthyma in species other than sheep and goats would be informative, but this should not be considered high priority research.

Group Discussion:

How can contagious ecthyma be controlled in susceptible animals?

A vaccine for domestic sheep is conmercially available. This vaccine probably would offer protection for susceptible game animals; however, the vaccine has not been tested or approved for use in these species. The vaccine is a live-virus vaccine which means that accidental establishment of the disease and its transmission could be a problem in sone species.

Besides members of the sheep family, what other species could be affected by contagious ecthyma?

Cases of contagious ecthyma have been reported in a number of species but it usually just causes localized mild lesions. The virus could potentially cause serious damage to elk and deer velvet. Remember that contagious ecthyma can cause painful localized infections in humans and animals suspected of having contagious ecthyma should be handled with care.

Why is there so little information on the affects of contagious ecthyna?

Contagious ecthyna is not considered to be a serious pathogen of game animals and thus little research has been conducted on it's affects.

Chronic Wasting Disease

Thorne/Wiliams

Species:

Mule deer, mule deer x white-tailed deer hybrids, elk

Agent:

Unknown, "slow virus"

Scientific And Common Names:

chronic wasting disease; slow virus disease

Known Geographic Distribution:

Colorado, Wyoming, and Ontario

Clinical Signs:

The primary clinical signs of chronic wasting disease are loss of body condition to a state of emaciation, behavioral alterations, excessive drinking, salivation and urination. Behavioral changes are usually very subtle early in the disease and may only be recognized by animal caretakers familiar with the individual animal. The animals may stay apart from the herd more than usual, they may be hyperactive including persistent pacing or repetitive motions, and they have unusual facial expressions which include droopy ears and a blank stare. The animals react to noise and can see. Tremors and ataxia are rare. Affected animals drink excessive amounts of water and late in the disease are often found at the waterer. They may have problems swallowing and they put their noses deep into the water even up to the level of their eyes. Excessive salivation and grinding of teeth is common. Affected animals have all been 18 months or older. Morbidity at any one time is usually low but mortality is 100%. In one facility in Colorado, 90% of deer held for 2 years or more developed the disease.

Gross Pathological Changes:

Emaciation is the most consistent gross alteration. Many animals have watery green rumen contents due to excessive ingestion of water and urine is very dilute. Aspiration pneumonia is seen in a few animals.

Microscopic Changes:

The only significant microscopic lesions are found in the brain and are characterized by intraneuronal vacuolation; astrocytosis, and spongiosis of the neuropil. Lesions are most severe in the olfactory tubercle, the hypothalamus, and the dorsal motor nucleus of the vagus. There is no inflammatory reaction.

Methods of Diagnosis:

Clinical signs are suggestive of chronic wasting disease, however, other diseases of the central nervous system may cause similar signs. Currently, the only method of diagnosis is by microscopic examination of the brain. Incubation periods are very long (years) and there is no way to test animals for this disease prior to the time they develop clinical disease.

Treatment:

There is no treatment for chronic wasting disease and it is apparently 100% fatal once clinical disease is recognized. Control in several facilities has been by depopulation of all cervids. Facilities with chronic wasting disease should restrict movement of animals to other facilities.

Other Species Affected:

Chronic wasting disease is very similar, and possibly the same as scrapie in domestic sheep and goats. Similar diseases are transmissible mink encephalopathy in mink and bovine spongiform encephalopathy. a recently recognized in cattle in England. Two rare human diseases. kuru and Creutzfeld-Jakob disease. are also considered to be related. Because the infectious agent causing these diseases has not been isolated, the exact relationship between these diseases has not been determined.

Clinical Signs In Additional Species:

Chronic wasting disease has only been identified in deer and elk.

Prevalence In Wild And Domestic Species:

Chronic wasting disease has only been recognized in a few facilities in Colorado, Wyoming and Ontario. A few suspect cases have been identified in free-ranging animals.

Importance To Domestic Species:

Chronic wasting disease has not been reported in domestic species. The relationship to scrapie is not known at the present time.

Transmission:

The mode of transmission is not known, but epidemiological investigations suggest that lateral transmission occurs.

Public Health Implications:

It is not known if humans can become infected with chronic wasting disease. Because several human diseases are similar to chronic wasting diseases it seems prudent to treat animals with chronic wasting disease and their tissues with care until we know more about the disease. Animals with chronic wasting disease should not be consumed by humans.

Research Needs:

Prevalence, occurrence of the disease in freeranging animals, relationship to other slow virus diseases.

Swine Erysipelas

Thorne/Williams

Species:

Swine

Scientific And Common Names:

Swine erysipelas, diamond skin disease

Known Geographic Distribution

Erysipelas may occur wherever domestic swine and domestic sheep are raised and where rodents are common.

Clinical Signs:

Effects of erysipelas in ferral swine are probably similar to those seen in domestic swine. The acute disease results in prostration, conjunctivitis, vomiting in some animals, and death. Red or purple patches may be present on the skin of the ears, abdomen, and insides of the legs. Milder cases are characterized by urticarial skin lesions that initially are reddish or purple diamond shaped blotches; these skin lesions occur primarily on the abdomen. The diamond skin lesions later become necrotic, and the skin dries into dense scabs. The chronic disease results in vegetative valvular endocarditis, which ultimately leads to sudden death.

Erysipelas also takes an arthritic form in older animals. Affected animals become lame as joints become enlarged and painful. Lameness may also be caused by laminitio.

Gross Pathological Changes:

Diamond shaped skin lesions may be present. The spleen and lymph nodes are enlarged and the mucosa of the stomach and intestine is inflamed, hemorrhagic, and possibly, ulcerated in animals that die in acute stages of the disease. Vegetative endocarditis occurs on heart valves, particularly themitral valve, in swine that live long enough to develop chronic lesions. Arthritic lesions are most common in limb joints and consist of hyperemia and hypertrophy of synovial membranes and villous projections extending onto joint cartilage.

Microscopic Changes:

Grossly apparent diamond shaped lesions in the skin are associated with thrombosis and inflammation of underlying small arterioles. Neutrophils predominate in the imflammatory reaction and bacteria are often numerous lesions in internal organs reflect a septicemia and are characterized by petechial hemorrhages, especially in the kidney, and focal fibrinoid necrosis of capillary tufts in glomeruli. Necrosis and thrombosis may occur in synovial arterioles. Septic emboli with neutrophilic excreation are common in small vessels of the brain, spinal cord, and choroid and ciliary process in the eye.

In chronic cases, mitral endocarditis characterized by vegetative lesions, thrombus formationand the presence of masses of bacteria. Congestive heart failure and splenic and hepatic infarction may occur. Villous synovial membrane hyperplasia, chronic nonsuppurative inflammation, and pannus formation are present in chronic cases of polyarthritis due to **E. rhusiopathiae**.

Methods Of Diagnosis:

In swine, erysipelas must be distinguished from hog cholera which may be difficult even for experienced diagnosticians. The spleen is usually enlarged, firm, and dark red with erysipelas, and the stomach is frequently highly inflammed. Valvular endocarditis is a common lesion seen in chronically affected swine. Arthritic lesions consist of enlarged joints, hypertrophied synovial linings, villous projections, erosions of cartilage, and greatly increased synovial fluid that is not grossly purulent.

Blood of swine with erysipelas should be cultured for **Erysipelothrix rhusiopathiae** complement fixation and agglutination tests can be used for serologic diagnosis, however the agglutination test may not always detect old infections.

Treatment:

Erysipelothrix rhusiopathiae is susceptible to penicillin G and ampicillin. Treatment should be accompanied by thorough disinfection of the affected animal's quarters and isolation from other animals.

Other Species Affected:

Many wild avian and mammalian species, especially rodents, small carnivores, pinnipeds, and cetaceans are susceptible to erysipelas. Infections in wild cervids and bovids are rare.

Clinical Signs In Additional Species:

Erysipelas is usually mild in domestic cattle and sheep, and the most common clinical sign in these species is arthritis and lameness. Presumably, captive wild bovids and sheep also would experience arthritis. Those animals that die exhibit generalized signs associated with septicemia.

Many species of birds are susceptible, and many experience fatal infection with turkeys being among the most severely affected. Blue comb may occur as a sign of cyanotic skin. Males may experience more mortality than females.

Prevalence in Wild And Domestic Species:

Erysipelas may be common where domestic swine and sheep are intensively managed or rodents are numerous. Erysipelas has not often been reported from free-ranging or captive wild ungulates, but it can be expected to occur more commonly as they are more intensively ranched and held in associated with domestic sheep and domestic or feral swine.

Importance To Domestic Species:

Erysipelas results in economic loss due to lameness and mortality when it occurs in swine and sheep. Losses of turkeys may be high; some outbreaks in turkeys have been associated with the presence of domestic sheep or sheep range.

Transmission:

Erysipelothrix rhusiopathiae is relatively resistant in the environment, and transmission is probably by ingestion or wound infection by contaminated material.

Public Health Implications:

The infection in humans is known as erysipeloid to distinguish it from human erysipelas, which is caused by a hemolytic **Streptococcus** species. Erysipeloid is usually manifested as i localized, painful, reddish, swollen wound infection; the wound usually is not suppurative. Arthritis occurs in nearby joints in about 5% of the patients, and septicemia is rare.

Research:

Additional knowledge regarding the effects of erysipelas in wild ungulates is needed and will become of increasing importance as they are more intensively ranched.

Group Discussion:

Under what conditions has erysipelas been a problem tc game ranchers?

Erysipelas has been diagnosed in ostrich and emu kept in a hog barn. Because of the potential problem of transmission of the bacteria from pigs to exotic birds, game ranchers should avoid allowing contact between these species or housing birds in places where hogs have been raised.

Rabies

Thorne/Williams

Species:

All mammals

Agent:

Viral

Scientific And Common Names:

Rabies, hydrophobia, rage

Known Geographical Distribution:

ubiquitous

Clinical Signs:

Rabies causes central nervous system signs in the form of abnormal behavior. Two syndromes termed "furious" and "dumb" rabies have been described; ruminants generally experience "dumb" rabies. Pharyngeal paralysis may result in altered vocalization and frothing of the mouth due to inability to swallow. Rabid cattle are frequently thought to have pharyngeal obstruction; presumably, the same misdiagnosis could occur with ranched wild ungulates. Affected animals may exhibit incoordination, tremors, and convulsions. Clinicals signs in a pronghorn antelope occurred 33 days after exposure and consisted of lethargy and loss of appetite followed in two more days by death. A black-tailed deer lost interest in moving about and showed signs of incoordination and posterior paralysis; it made agonizing bellows when approached by humans. A moose displayed clinical signs of outhriftiness, circling, possible blindness, and intermittent aggression.

Gross Pathological Changes:

None. Behavioral abberations may result in incidental phathologic findings such as presence of porcupine quills, inappropriate ingesta, and traumatic damage to the mouth or body.

Microscopic Changes:

Nonsuppurative encephalitis and the presence of neuronal inclusion bodies, termed Negri bodies are the most common microscopic changes. Negri body-like inclusions may occur in normal moose.

Methods Of Diagnosis:

Wild animals suspected of having rabies should never be held for observation; they should be euthanized and submitted for necropsy. They must not be shot or struck in the head because examination of the brain is required for diagnosis of rabies. If the entire carcass cannot be submitted for necropsy, the head should be removed; sealed in a dry, leak-proof container; cooled with ice and kept cold; and delivered as quickly as possible to a laboratory equipped to diagnose rabies. Consultation with the diagnostic laboratory should be made prior to shipment. Diagnosis of rabies requires demonstration of the virus. Fluorescent antibody techniques are most commonly used to diagnose rabies and are rapid and reliable. Brain material is used for fluorescent-antibody tests. Innoculation of suspect brain material into the brain of 3- to 4-week old mice may also be used as a diagnostic technique, especially when human exposure has occurred. Although microscopic examination of the brain to detect Negri bodies is no longer frequently used, microscopic examination of the brain occasionally detects the presence of nonsupurative encephalitis and Negri bodies in rabid wild animals when rabies originally was not considered in the diagnosis. Gross and microscopic examination of brains from animals suspected of having rabies may result in diagnosis ofother disease conditions responsible for the clinical signs.

Treatment:

There is no method of treatment for rabies; animals suspected of being exposed to rabies or having rabies should be destroyed. There is no approved or proven reliable vaccine available for use in wild animals. Research is currently underway to test inactivated rabies vaccines.

Other Species Affected:

All mammals are susceptible to rabies. It is uncommon in wild cervids and bovids, but rabies is more common in wild and domestic carnivores. Skunks and some species of bats may experience prolonged course of infection during which rabies may be transmitted without overt display of signs of disease.

Clinical Signs In Additional Species:

Signs of rabies are similar in all wild cervids and bovids. Carnivores more frequently experience "furious" rabies by displaying aggressive behavior. Abnormal behavior in any wild animal should be considered as a possible indication of rabies.

Prevalence In Wild And Domestic Species:

Rabies is now more prevalent in wild species than in domestic animals, but it is rare in wild cervids and bovids.

Importance To Domestic Species:

When it occurs, rabies is important to both large and small domestic species because it is always fatal and because of its importance of a zoonotic disease. The occurrence of rabies in domestic livestock is almost always due to a bite of an infected carnivore. Rabies in domestic livestock is usually a sporadic disease and rarely occurs as an epizootic disease.

Transmission:

Inoculation by bite or wound contamination of virus laden saliva of an infected animal is the most important means of transmission. Rabies virus may be transmitted in the saliva of an infected animal before it displays clinical signs of the disease. No case of rabies has ever been traced to exposure to a wild deer. Skinning, dressing, and preparation of meat of wild cervids and bovids should not be considered as exposure to rabies.

Public Health Implications:

Rabies is essentially 100 percent fatal in humans and is, therefore, highly feared. Pre- and postexposure immunoprophylactic techniques are available. Ordinarily, work with captive wild cervids and bovids should not warrant preexposure prophylactic measures.

Group Discussion:

What should be done if you suspected a game animal of having rabies?

Because of the serious implications of having a rabid animal in contact with humans and other animals, the animal showing nervous signs suggestive of rabies should be immediately euthanized. The carcass should be tested for rabies and other diseases which could cause nervous signs.

What can game farmers do to control rabies?

Game ranchers should educate themselves about the presence of rabies in their area and the species most often affected. Wild animals showing signs suggestive of rabies should be reported to the appropriate authorities. Precautions should be made to keep these wild animals away from game farm animals. All pet animals (dogs and cats) on the farm should be vaccinated for rabies . Rabies vaccines are not currently available for game animals.

What wild species are most important in spreading rabies?

Depending upon the area, skunks and foxes are the species most frequently affected by rabies. Raccoons may also contract rabies and be an indicator of rabies in a particular area.

Summary

Wildlife disease experts were contracted to survey the disease status of game animals of North America. In consultation with the Western College of Veterinary Medicine a list of diseases of concern to Saskatchewan was compiled and diseases were assigned to consultants based on their areas of expertise. To ensure that all areas of North America were covered, four internationally known wildlife disease experts were contracted, based on their geographical location.

Consultants made formal presentations on each assigned disease at a two-day conference in Saskatoon.

Discussions following each presentation helped to:

1) more precisely define the known geographical distribution of the disease;

2) assess current testing and treatment regimens;

3) assess the risk or impact on game animal and wildlife populations if a new disease was accidentally introduced to Saskatchewan;

4) identify additional research required for the development of new or refinement of existing disease testing protocols for importing game animals; and

5) priorize identified future game animal research needs.

Paraelaphostronqvlus tenuis, commonly known as brain worm or meningeal worm, was concluded to present the greatest threat to game farm animals and provincial wildlife populations if accidentally introduced to Saskatchewan. Wildlife disease experts agreed that **P. tenuis** was the highest priorety in future wildlife disease research, in view of current animal translocations among game farms. In particular, the timing and frequency of the current fecal testing procedure should be re-evaluated on the basis of research studies now underway.

In addition to **P. tenuis**, the conference identified an additional five diseases of immediate concern to Saskatchewan. Two of these diseases, Eleophorosis and Besnoitiosis are not present in Saskatchewan, but would have devastating effects on game farm herds and wildlife populations if accidentally introduced to the province. Until fail-safe diagnostic procedures are available, importation of game species that are normal hosts of these diseases is not recommended.

Johnes disease, chronic wasting disease and Malignant Catharrhal Fever are three additional diseases of immediate concern to game farmers. These diseases occur in Saskatchewan, but would have devastating financial consequences if introduced to game farms as no reliable treatments are available. These diseases do not pose a major threat to free ranging wildlife populations.

The other diseases adressed in this survey were considered to be of minor concern to game farming and wildlife management interests. In general, these diseases were concluded to:

1) have little potential for economic impacts on the game farming industry;

2) have adequate treatment procedures available;

3) be controllable through husbandry practises;

4) be endemic in wildlife populations; or

5) have low potential for transmission from game farms to wildlife populations.

The diseases considered to be of minor concern include;

Anaplasmosis

Contagious Echthyma

Ectoparasites

Erysipelas

Leptospirosis

Pseudorabies

Q-Fever

Rabies

Yersiniosis

Lyme disease

"Other Nematodes"

In addition to these specific conclusions, the conference identified a series of general recommendations to refine prevention and control of game animal diseases. These include:

1) Veterinarian's reports should be required to substantiate the occurance of named diseases.

   This is particularly important in the case of diseases where occurance triggers drastic control measures, de-population and restriction on herd movements.

2) A general advisory should be issued to all game farmers warning that game farms should not be established in close proximity or association with sheep or swine operations.

   The rationale for the advisory is that there is much greater risk of accidental infection of game animals (with sheep- associated Malignant Catharrhal Fever, Erysipelas, Pseudorabies, and contagious ecthyma) and domestic livestock (with flukes and Elaeophora).

3) An information system should be established to pool the knowledge of established game farmers.

   The rationale for this recommendation is that some disease problems can be effectively managed through husbandry practises (eg. Yersiniosis control by animal stress reduction).

4) Education bulletins should be issued to game farmers publicizing the potential risks of conventional livestock diease in the game farm situation.

   The rationale for this recommendation lies in the potential problems arising from ignorance of livestock diseases such as Erysipelas.

5) The carcass of any game animal that dies should be subjected to a thorough pathalogical examination by qualified diagnosticians.

   Although this provision exists in the current requirements of **The Game Farming and Game Products merhandising Regulations**, its vital importance to game farmers must be emphasized. The potential hazard of disease spread and economic losses to both game farmers and conventional livestock producers is too great to not enforce this requirement stringently.

6) Any pets held on game farms should be vaccinated against rabies.

7) This disease survey should be reproduced as a bulletin and distributed to all government and non-government wildlife agencies and government agencies having jurisdiction over game farming.

8) Disease testing protocols for import of game animals developed based on information in this survey should be distributed to appropriate government agencies in North America. Other North American government agencies should be encouraged to develop and distribute protocols for importing game animals into their jurisdiction.

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